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JOHN WESLEY BOVÉE
1861-1927

The American Journal of Obstetrics and Gynecology

VOL. XIV ST. LOUIS, OCTOBER, 1927 No. 4

IN MEMORIAM

JOHN WESLEY BOVÉE

A MEMBER OF THE ADVISORY EDITORIAL BOARD OF THIS JOURNAL

DR. JOHN WESLEY BOVÉE, a Fellow of the American Gynecological Society since 1897, died at the Columbia Hospital for Women in Washington, D. C., on September 4, 1927. He was born in Clayton, N. Y., in 1861, the son of William Henry and Sarah Elizabeth Bovée. His early education was received from private tutors and in the high schools of Dexter and Chaumont, N. Y. He graduated with the degree of M.D. in 1885 from Columbian University, now George Washington University, in Washington, D. C. He was Gynecologist to the Columbia Hospital from 1890 to the time of his death; to the Providence Hospital from 1891 to 1908; and to the George Washington Hospital from 1899 to 1927. For many years Dr. Bovée was Professor of Gynecology at George Washington University, resigning the position in 1926. An active member of the American Medical Association, he served as Chairman of its Section on Gynecology and Obstetrics in 1907. He was a member and past president of the Southern Surgical Society, a Fellow of the American College of Surgeons and one of its founders, a member and former president of the American Gynecological Society. Dr. Bovée was a prolific writer on subjects pertaining to his specialty and his contributions to medical literature numbered more than 200 papers. He edited and wrote several chapters in his most notable work, Bovée's *Practice of Gynecology*.

Dr. Bovée was essentially a self-made man. His native ability, industry, and ambition to excel finally made him an authority in gynecology and brought to him many well-deserved honors. He was especially fond of teaching, not only of undergraduate students, but also hospital interns and nurses, and spent many hours going over and correcting hospital histories and charts. He zealously guarded his rights as a physician and member of hospital medical boards. He had been twice married but left no family. Dr. Bovée was likewise a member of the American Gynecological Club and of the Cosmos and University Clubs of Washington.

By his death the American Gynecological Society loses one of its oldest and most useful members and our country a citizen of note, who was ever ready to sacrifice his leisure and convenience to serve his country, his profession, and his fellowmen.

G. BROWN MILLER.

President's Address

THE SPIRIT OF SERVICE

By Arthur H. Curtis, M.D., Chicago, Ill.

Presidential Address at the Fifty-second Annual Meeting of the American Gynecological Society Held in Hot Springs, Va., May 23-25, 1927

THE subject of these brief remarks may be somewhat too delicate for one not gifted in the art of discourse. If so, I beg of all of you a kind indulgence and will proceed at once with what I have to offer.

Others, in former years, have presented surveys of the work produced by this Society. At the most, reiteration of the salient features of some of those presidential addresses is all that you will care for at this time.

Our keen-minded, truth-loving Whitridge Williams pictured, most strikingly, the part which we had played in the advancement of obstetrics; in summary, a truly insignificant and wholly unworthy list of papers, emanating from a group of men supposed to be the leaders in this field.

In contrast, the following year, after most painstaking and thorough study, Dr. Watkins revealed to us a large group of valuable contributions in the field of gynecology. Many of these were epoch-making papers which will continue to have intrinsic worth long after we are gone.

It remained for Dr. Chipman, of Montreal, to complete the survey; to make us proud of our past, yet aware of our faults, and to inspire us with ambition for future progress. Surely no one who heard that address could fail to become imbued with more lofty ideals and a purer spirit of love for his confrères and sympathy for suffering womankind.

Turning to the present, it may be well to note wherein we should do better. One of our Fellows recently deplored the lack of younger members, the limited number who attend our meetings, and the notable absence of the spirited debate and controversy which should mark these gatherings. He is convinced that every one who differs with the author of a paper should enter the discussion; that silence means agreement, and that nowadays there tends to appear in our transactions much that creates a wrong impression because undue politeness and over-zealous courtesy inhibit refutation of erroneous views. As another expressed it: "We have too much back-slapping and back-scratching —too much golf and other play at meetings; there is a lack of scientific work and a want of intelligent discussion because our members fail to study and produce."

I most warmly favor, in this Society, the maintenance of a small group, selected chiefly for achievement and ability. Small membership fosters freedom of discussion and more intimate personal contact, so helpful to us all. From every annual meeting I return to duties at home, warmed by increasing friendship for those whom I have seen once more, keenly ambitious to do better work, inspired with higher ideals and imbued with a desire to be of utmost service to my patients and to the younger generation who look to us for guidance.

Does spirited debate and argument achieve so much? If so, then why, in consultation, where we give our best, do we not argue heatedly, rather than decide by sympathetic interchange of views and careful logic, the plan best suited in the management of those intrusted to our care? Likewise, in the laboratory, where growth of thought and interchange of views are at their highest level, is not our progress greatest when the talk is least and arguments are few?

Adequate discussions are vital. Differences of opinion should be freely aired. But I have always held that one should feel a mission when he rises to his feet; at least that he should be impelled to speak because there still remains something of special import left unsaid.

Just as we find that many roads lead to Rome, so each one has his way. Some men talk often and say much. Others are silent, and, for most of us, silence is golden. It is not requisite that we refute false views; they die a natural death and suffer less from agony if spared the useless stimulation of protracted argument.

One person among us illustrates some points that I would make. Almost unrivalled in creative work in all the history of our specialty, he stands out even more preeminent as one whose greatest interest centers solely in a search for truth. He is above all thought of controversy and listens earnestly, attentively, without retort, to every contrary view expressed. Discussion means to him a sacred consultation, not an altercation, a conference of trained minds in a conjoined effort to disclose new facts which lead to progress. We honor him for his work, we admire him for his modesty and gentle spirit, and we think not a bit less of him because he sits in Sphinx-like silence throughout the greater portion of our meetings.

All told, we make some progress. And, speaking as your former secretary, I wish to express publicly my admiration for the high-minded cooperation and spirit of service so generally in evidence among our members. This Society is, seemingly, now at its best in helpfulness to the profession at large and in productive scientific work.

Also, let us not be too oblivious of the service rendered by our individual Fellows in their tasks at home. In New York City, at the Woman's Hospital, there has grown up an institution so well organized

that it is now without a peer in excellence of management. In systematic after-care of patients, in conference among the members of its staff, in accuracy of records and statistics, it is a model of which we may well be proud.

We number also, here, a leader in the fight for control of cancer; likewise, one who has done more than any other man to elevate the standards of our hospitals; also, the authors of our best known standard textbooks, and the founders and editors of journals recognized as excellent throughout the world of medicine.

Then, too, we have a larger group; these are thoughtful teachers of young men, leaders, almost without exception, in our greatest schools. Vitally interested in their work at home, mindful of the welfare of their patients, their service will be long remembered and their influence felt throughout the years to come. And so we might go on, with more than kindly words about all gathered here. But time is short, and my aim should not be to discourse to the end about all of your virtues.

Now let us turn, for a brief space, to thoughts of those whose seats are vacant. As we look back upon quite recent days spent with our former Fellows, now deceased, no conference is required to help decide wherein they rendered service. The influence of their personal lives is ever with us, a permanent and constant heritage; a gift more rare, to me, than all their scientific work. Extended backward to us, from the life beyond, we feel their guiding hands put forth to lead us.

In my own case, I sense, with special force, the helpfulness of a most modest, truth-inviting man. Each day he leads me on, through impress of the life he lived. Despite a generous group of scientific contributions, to me he left much more in demonstration of a humble spirit, in his most patient care to risk no life in needless surgery, in precept that to cheer with helpful word and kindly smile is of the utmost value. We need these lessons in the art of kindliness; throughout our busy lives we should all nurse our sympathy for those who suffer; we need, also, to learn to be more generous to our young colleagues, just as this man was, in countless ways, without a vestige of that common fear that some day they might grow to be competitors.

This word of retrospect stirs up fond memories of others gone, and from their lives we learn a better regulation and adjustment of our own. We learn the fruitlessness of uncurbed personal ambition, of false desire for unearned praise and glory, of the too great price which money sometimes costs, of heartaches caused by selfishness where kindness should be found.

A final thought, in closing. It is a custom of our race to be forever striving,—in this country striving to our utmost. I wonder whether we have not engulfed ourselves in an American treadmill

habit of continual and untiring effort to the exclusion of all else that makes for happiness? The public utterances of those in medicine whom we respect center about the need for *progress,* about *work* poorly done or left undone, about our misdirected energies, and how we might accomplish more. This *urge to do* applies with added force to those who practice in our specialty, and in our double-specialty perhaps the most of all. What is it all about? In serving, sacrifice comes first, but let us also serve our families and ourselves. More respite from unceasing toil should be the share of each and all of us. Otherwise the truly perfect spirit of service will never be fulfilled.

Original Communications*

PERITONEAL ENDOMETRIOSIS DUE TO THE MENSTRUAL DISSEMINATION OF ENDOMETRIAL TISSUE INTO THE PERITONEAL CAVITY

By John A. Sampson, M.D., Albany, N. Y.

(From the Gynecologic and Pathologic Departments of the Albany Hospital and the Albany Medical College)

AT THE meeting of the American Gynecological Society in 1921, the writer presented a paper[1] on perforating hemorrhagic cysts of the ovary and their relation to pelvic adenomas of endometrial type. Twenty-three of these cysts were reported at that time. In fourteen of them, a histologic study had been made of the peritoneal lesions, apparently resulting from the escape of the contents of the cysts into the peritoneal cavity. Endometrial tissue was found in thirteen. I concluded that the hemorrhagic ovarian cysts, associated with the peritoneal lesions containing endometrial tissue, were endometrial cysts filled with menstrual blood and that the peritoneal endometriosis arose from the escape of the cyst contents into the pelvis and the resulting peritoneal reaction. In view of the theories which have arisen to explain the origin of the peritoneal endometriosis associated with these cysts, the following quotation from that paper may be of interest.

> The question naturally arises: in what way do the contents of the cyst or ovary cause the development of these adenomas? Is it due to some specific irritant present in the cyst contents which stimulates the peritoneal endothelium to a metaplasia with the development of endometrial tissue typical both in structure and function? Some may assert that dormant endometrial epithelium may be present in the tissues soiled by the contents of the cyst and this is stimulated to further growth. It seems to me that the conditions found in many of these specimens are analogous to the implantation of ovarian papilloma or cancer on the peritoneal surfaces of the pelvis from the rupture of an ovarian tumor containing these growths.

At that time I believed that the ovary was the principal, if not the only, source of the peritoneal implantations which arose from endometrial tissue disseminated by the menstrual perforation of an endometrial cyst or by the menstrual reaction of endometrial tissue on the surface of that organ. Up to that time only a few peritoneal lesions had been observed by me without demonstrable endometrial tissue in the ovary. An attempt was made to explain these on the basis that they had arisen from the implantations of endometrial tissue, derived from small

*The current issue of the Journal is devoted to the first part of the Transactions of the Fifty-second Annual Meeting of the American Gynecological Society, held at Hot Springs, Va., May 23, 24, 25, 1927. The discussions on the papers presented will be incorporated in part in the next issue of the Journal.

Note: The Editor accepts no responsibility for the views and statements of authors as published in their "Original Communications."

endometrial foci in the ovary, and that the latter had either been destroyed by menstrual reactions or else were so small that they had been overlooked.

Later studies convinced me that peritoneal endometriosis frequently occurred without endometrial tissue in the ovaries, and some other way by which this tissue might reach the peritoneal cavity was sought. Blood was observed escaping through the abdominal ostia of the tubes of patients operated upon during menstruation. Bits of endometrial tissue were found in the lumina of some of these tubes. The frequent close anatomic relation between the distribution of the peritoneal lesions and the fimbriated ends of the tubes was observed at operation. It was found that the tubes were usually patent in patients with peritoneal

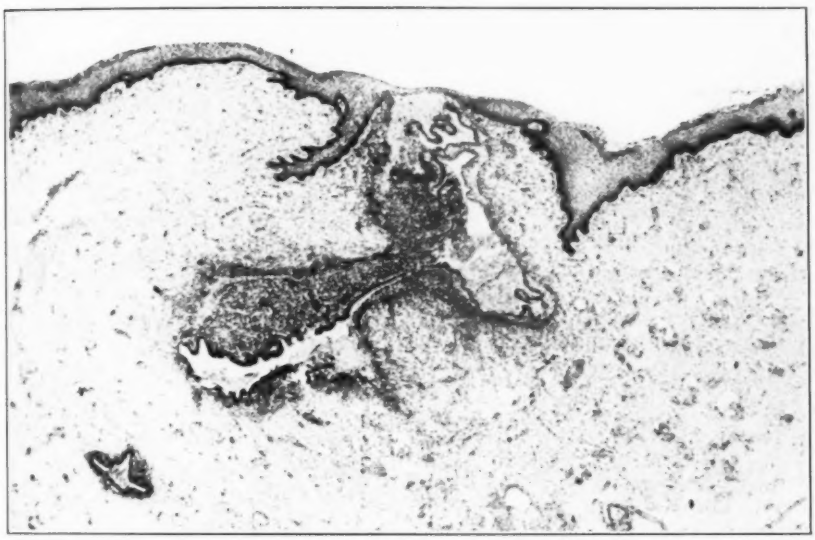


Fig. 1.—Photomicrograph (x 25) of a portion of the posterior vaginal wall with a submucous endometrial cavity about to rupture into the vagina (Case 4 of previous article[2]). The patient had peritoneal endometriosis fusing the anterior surface of the uterus with the bladder, and a similar lesion obliterating the bottom of the posterior culdesac with invasion of the rectosigmoid and the posterior vaginal wall. The cavity contains blood and bits of endometrial tissue; operation on the second day of menstruation. The vaginal mucosa over the cavity is very thin and had the operation been deferred subsequent menstruations of the endometrial tissue lining this cavity undoubtedly would lead to a rupture through the attenuated vaginal mucosa with the discharge of the menstrual contents of the cavity into the vagina. The actual rupture of two similar cavities of the vaginal wall into veins was observed in this case and embolic implantations of endometrial tissue in near-by vessels was found (Figs. 58, 59, 60, 61 and 66 of previous article[2]). It is conceivable that similar endometrial cavities situated in the ovary or in any other pelvic structure, whose endometrial lining reacted to menstruation, might rupture and discharge their contents into the peritoneal cavity and also that implantations of endometrial tissue might occur on the peritoneum just as they occurred in the veins of this patient.

endometriosis. All these data, together with the indication that, at times, endometrial tissue escaped into the peritoneal cavity from other sources than menstruating tissue in the ovary, pointed to the backflow of menstrual blood from the uterine cavity through the tubes as one of these sources and also to epithelium escaping from the tubal mucosa as another. The purpose of this paper is to present the evidence indicat-

ing the origin of peritoneal endometriosis from the implantation of endometrial tissue disseminated by menstruation. Its origin from other sources will be considered in a later article.

If it is true that peritoneal endometriosis arises from the implantation of endometrial tissue carried by menstrual blood escaping from endometrial foci in the ovary and also from the uterine cavity through the tubes, the following three suggestions or theories present themselves.

1. Peritoneal endometriosis might be caused by menstrual blood escaping from endometrial tissue on or in other pelvic organs and structures than the ovary.

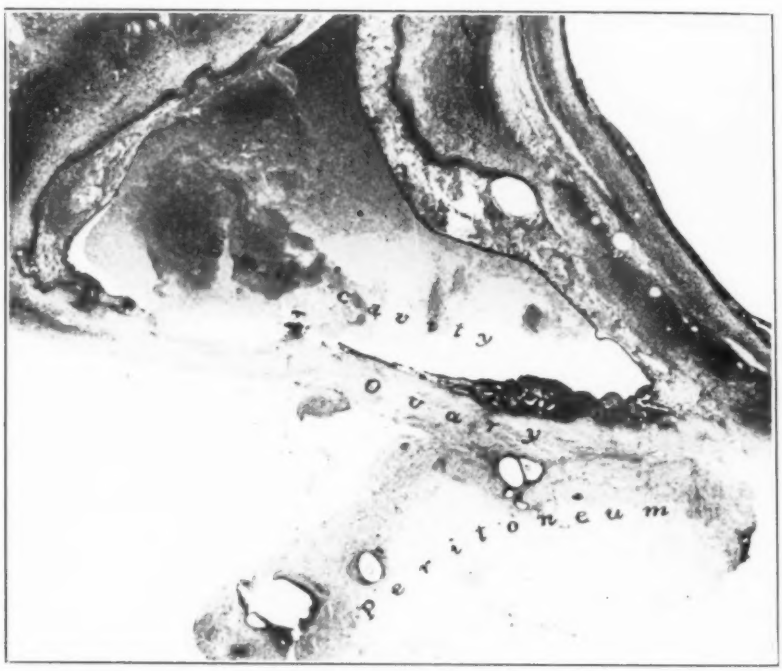


Fig. 2.—Photomicrograph (x 10) of a portion of the ovary adherent to the posterior layer of the broad ligament. Uterus, right tube, and ovary removed for a large leiomyoma on the second day of menstruation (Case 1 of previous article[2]). The ovary was firmly adherent by its *lateral surface* to the posterior layer of the broad ligament. Peritoneal endometriosis was present in the structures about the ovary and in the posterior culdesac. The right tube and ovary, together with the portion of the peritoneum attached to the latter, were carefully removed and placed in formalin before attempting to remove the uterus. A typical endometrial cyst or cavity, filled with blood, is shown and also endometrial lesions in the peritoneum adherent to the surface of the ovary. The endometrial tissue lining the cyst and that in the peritoneum must have had a common origin or one was secondary to the other. Could the peritoneal endometriosis possibly have arisen from the escape of the contents of the endometrial cyst into the pelvis?

2. Endometrial tissue in the ovary might arise from the implantation of bits of that tissue carried by menstrual blood escaping through the tubes and also from endometrial foci on other pelvic organs or structures.

3. The peritoneal endometriosis, so often associated with endometrial tissue in the ovaries and apparently secondary to the latter, at times, might have arisen from some other source.

I greatly appreciate the interest shown by others in those observations and theories and relish most of all their objections and criticisms. The latter are stimulating and will make for a better understanding of the entire subject. I fully realize that the implantation theory does not account for all instances of ectopic endometrium-like tissue in the pelvis and that menstruation is only one means of disseminating that tissue. I value the opinion of those who believe in the differentiation of celomic epithelium as a source of endometrial tissue in the ovary and peritoneum. In spite of the increasing acceptance of the serosal or celomic epithelial origin of peritoneal endometriosis, I believe that bits of endometrial tissue, carried by menstrual blood escaping into the peritoneal cavity, at times, become implanted on the peritoneum and thereby constitute one source of peritoneal endometriosis, just as the implantation of cancer cells on the peritoneum constitutes one cause of peritoneal carcinosis. I

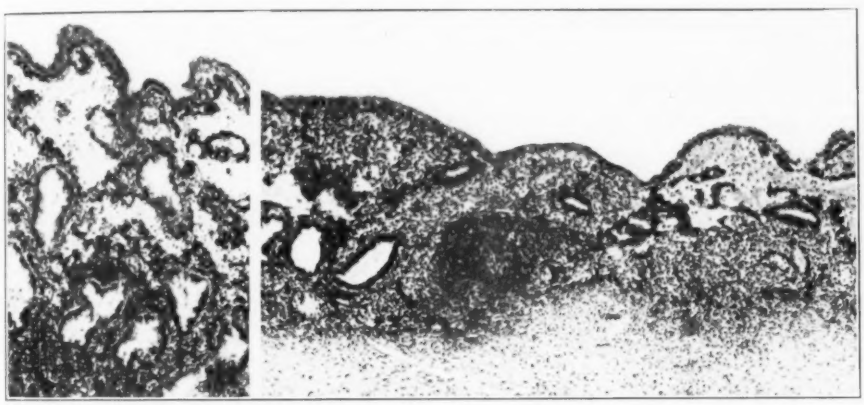


Fig. 3.—Two photomicrographs (x 60), the first of a portion of the menstruating mucosa lining the uterine cavity and the second of the endometrial lining of the cyst shown in Fig. 2. Both show the same reaction to menstruation.

believe that menstrual blood escapes into the peritoneal cavity from the following sources:

1. The menstrual rupture or perforation of the wall of an endometrial cavity (cyst), most frequently seen in the ovary.

2. The menstrual reaction of endometrial tissue, growing on the peritoneal surface of the ovary or any other pelvic organ or structure.

3. A backflow, through the tubes, from the uterine cavity and also from the menstrual reaction of the tubal mucosa.

All phases of this theory, as to the implantation on the peritoneum of endometrial and tubal tissue disseminated by menstruation, must be considered seriously, if each of the following statements can be shown to be true or even possibly true.

1. Whenever endometrial tissue reacts to menstruation this reaction is the same whether in that lining the uterine cavity or situated at a dis-

tance from it, and, at times, bits of this tissue are set free in menstrual blood and may be carried (disseminated) by the latter from these foci.

2. Endometrial tissue, disseminated by menstruation, is sometimes alive and will continue to grow, if transferred to situations suited to its growth.

3. The peritoneum is suited to the growth of endometrial tissue.

4. Menstrual blood, carrying with it bits of endometrial tissue, at times, escapes from ectopic endometrial foci into the peritoneal cavity.

5. Occasionally menstrual blood escapes from the uterine cavity into the tubes. Bits of endometrial tissue have been found in blood in the lumina of the tubes of menstruating uteri. Sometimes the lumen of the

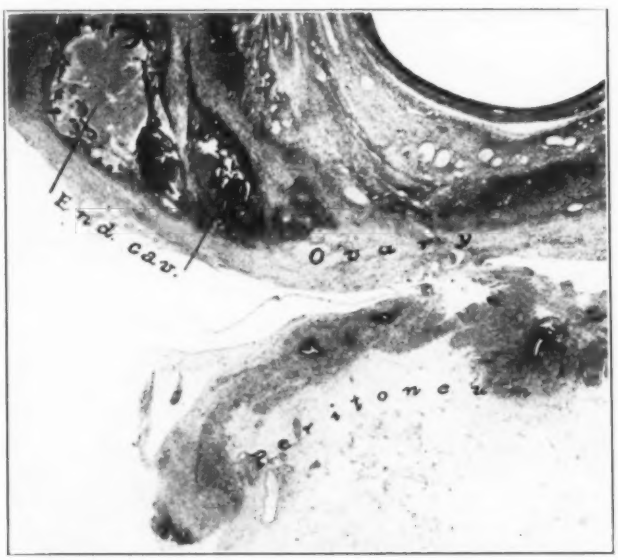


Fig. 4.—Photomicrograph (x 10) of a portion of the ovary and adherent peritoneum shown in Fig. 2 at another level. The endometrial cyst or cavity of the ovary contains blood with bits of endometrial tissue set free by the menstrual reaction of its lining, just as they are set free in menstrual blood arising from the mucosa lining the uterine cavity (see next illustration). Should such a cyst rupture during menstruation, its contents (blood and endometrial tissue) would escape into the peritoneal cavity and might cause adhesions. What would be the possible origin of any endometrial tissue in structures adherent to the ovary, such as the peritoneum shown in this illustration?

interstitial portion of the tube is of sufficient size to permit bits of endometrial tissue to pass through it.

6. The lesions of peritoneal endometriosis often occur in situations and under conditions indicating their origin from the above mentioned sources.

7. The local reaction of the peritoneum to the endometrial tissue in peritoneal endometriosis is similar to the local reaction of the peritoneum to the cancer in peritoneal carcinosis of implantation origin.

THE MENSTRUAL DISSEMINATION OF ENDOMETRIAL TISSUE

The histologic study of uteri, removed during the various stages of the menstrual cycle and in which the veins have been injected, demonstrates the mechanism by which endometrial tissue may be disseminated from that organ (See previous article).[2] The uterine mucosa contains venous capillaries which sometimes are dilated to form sinuses. These sinuses empty into similar sinuses (endothelial lined spaces without definite walls) of the myometrium and the latter empty into the arcuate veins which convey the venous blood from the uterine tissue into the venous circulation outside of that organ. During menstruation the venous capillaries of the mucosa rupture and blood escapes into the sur-

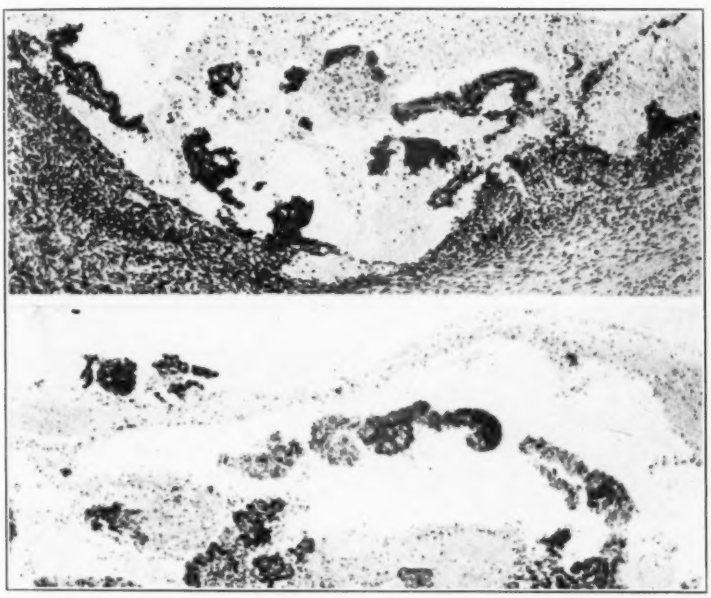


Fig. 5.—Two photomicrographs (x 60), the upper one of the menstrual contents of the endometrial cavity of the ovary shown in Fig. 4 and the other of the uterine cavity. They are much alike. Blood and fragments of endometrial tissue are present in both, histologically in a fair state of preservation and possibly as capable of living, if transferred to a suitable situation, as endometrial emboli disseminated into veins during menstruation.

rounding tissue and bits of the latter are set free in the extravasated blood. This blood breaks through the surface epithelium of the mucosa into the uterine cavity, often carrying with it bits of endometrial tissue suspended in that blood. These studies suggest that extravasated menstrual blood in the uterine mucosa, at times, might escape back into the lumen of the ruptured capillaries and sinuses from which it came and carry with it (disseminate) endometrial tissue into the venous circulation of the uterus. This actually occurs as has been shown.[2]

The histologic study of the endometrial tissue of a direct or primary endometriosis (so-called adenomyoma arising from the direct invasion of

the uterine wall by the mucosa lining its cavity) shows that this tissue contains venous capillaries (Fig. 12 of previous article[2]) similar to those of the mucosa lining the uterine cavity and that the reaction to menstruation of this misplaced endometrial tissue is similar to that of the former, except that it is not as constant or as general as that of the uterine mucosa. The endometrial tissue of a direct endometriosis, in its invasion of the myometrium often penetrates the spaces occupied by the

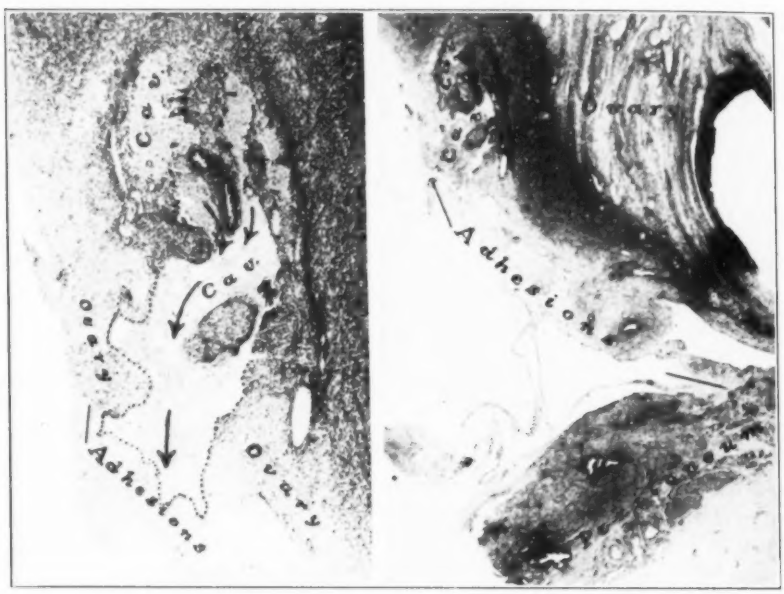


Fig. 6.—Two photomicrographs (x 25 and 10) of a portion of the right ovary and adherent peritoneum shown in the preceding illustrations, at another level. The endometrial cavity of the ovary appears much smaller and nearer the surface of the ovary. It is similar to the endometrial cavity of the vaginal wall shown in Fig. 1. Both patients were operated upon the second day of menstruation and both endometrial cavities contained blood and bits of endometrial tissue. The endometrial cavity of the vaginal wall was about to rupture and discharge its menstrual contents into the vagina. The endometrial cavity of the ovary had possibly ruptured into the peritoneal cavity at a previous menstruation and was closed by newly-formed connective tissue (adhesions) growing over it, as shown by the fibroblastic character of the adhesions covering the ragged rent in the wall of the cavity. The "rent" in the wall of the cavity and the inner surface of the adhesions covering it have been accentuated by dotted lines. These adhesions (see second photomicrograph) are continuous with similar adhesions uniting the lateral surface of the ovary to the posterior layer of the broad ligament. We have evidence that the adhesions uniting the ovary to the peritoneum might have arisen from the escape of the menstrual contents of the endometrial cavity of the ovary and that bits of endometrial tissue are often present in menstrual blood. Endometrial tissue is "embedded" in the peritoneum adherent to the ovary. Could there be any relation between the endometrial tissue escaping from the cavity of the ovary and that "embedded" in the peritoneum adherent to it?

vessels and sinuses of the uterine wall but is separated from the lumina of the latter by their endothelial lining. This has been emphasized by Robert Meyer[3] and Kitai in their descriptions of the relation of misplaced endometrial tissue to the lymphatics of the uterine wall. The study of specimens, in which the veins have been injected, has convinced me that the majority of the vessels of the uterine wall, which previously I had considered to be lymphatics, are venous sinuses. It might be as-

sumed that in the menstrual reaction of this misplaced endometrial tissue, bits of it might escape into its own venous capillaries and even into the lumen of a sinus of the uterine wall along which the endometrial tissue sometimes grows in an extra- or retroendothelial course. I have

Fig. 7.—Two photomicrographs, the first (x 10) of a portion of the posterior layer of the broad ligament adherent to the lateral surface of the ovary. The adhesions indicate that there had been a reaction to some irritant escaping into the peritoneal cavity. Fig. 6 indicates that blood might have escaped into the peritoneal cavity from the menstrual reaction of endometrial tissue in the ovary. A peritoneal endometriosis is present and if it were caused by the escape of menstrual blood from the ovary, the endometrial tissue of the endometriosis must have arisen, either from a metaplasia of the peritoneal mesothelium due to some specific stimulant in menstrual blood and not found in other well-known peritoneal irritants, or else from the actual implantation of bits of endometrial tissue often present in menstrual blood. There is a hyperplasia of the peritoneum about the endometrial foci. For a higher magnification of the endometrial "deposit" indicated by *a*, see the next photomicrograph.

The second photomicrograph (x 130) is of the endometrial lesion indicated by *a* of the preceding illustration. The tissues of the peritoneum are growing over (embedding) the endometrial tissue. The fibroblastic character of the peritoneum covering the endometrial tissue shows that it is of recent origin and histologically it is the same as that of the adhesions covering the rent in the wall of the ovarian endometrial cavity (Fig. 6). This suggests that possibly they are of the same age. The reaction of the peritoneum to the endometrial tissue in this instance is similar to that sometimes found in peritoneal carcinosis of implantation origin. I believe that we are justified in stating that peritoneal endometriosis, at times, arises from the implantation of endometrial tissue carried by menstrual blood escaping from endometrial foci in the ovary, and probably did in this instance. If so, some of the endometrial tissue, set free by menstruation, must have been alive.

not definitely demonstrated this in a careful study of many sections from many blocks of tissues from uteri with a direct endometriosis which were removed during menstruation, but I believe that either it has been seen by others or will be found. The same applies to misplaced endometrial tissue of other origin than a direct invasion of the uterine wall by its mucosa. In one instance of endometriosis of the culdesac, presenting in the posterior vaginal vault (a so-called adenomyoma of the rectovaginal septum) the actual escape of the menstrual contents of two ectopic endometrial cavities into adjacent veins was found. As a result of this observation I believe that a similar condition occasionally may arise in any situation, where endometrial tissue reacts to menstruation.

Since bits of the uterine mucosa, at times, escape into the venous circulation of the uterus during menstruation, certain questions arise. What is its pathologic and clinical significance? Could it possibly give rise

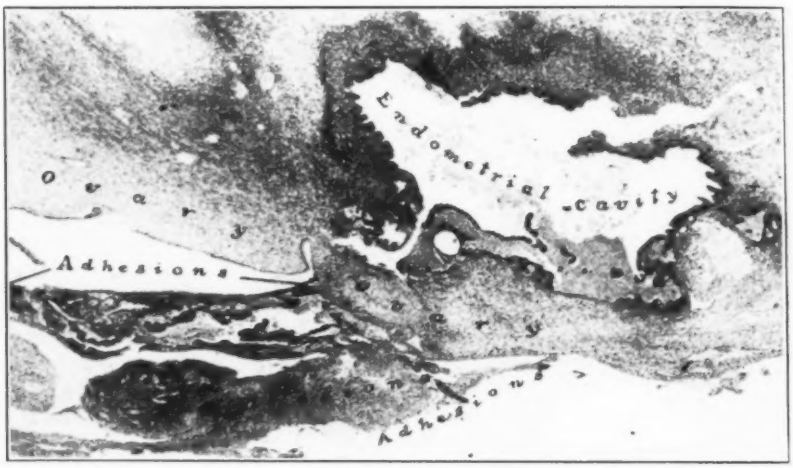


Fig. 8.—Photomicrograph (x 20) of the opposite end of the endometrial cavity shown in Fig 6. Endometrial tissue is enmeshed in adhesions on the surface of the ovary. Many sections were studied from this portion of the "block" and positive evidence of a perforation of the wall of the cavity was not found in this situation. If endometrial tissue had escaped from or had invaded the ovary at this place, the repair has been so complete that the portal of exit or entrance has been effaced.

to metastatic or embolic endometrial lesions not only in the uterine wall but also outside of that organ?

EVIDENCE OF THE VIABILITY OF ENDOMETRIAL TISSUE DISSEMINATED BY MENSTRUATION

If endometrial tissue disseminated by menstruation is "thrown off to die" and is either actually "dead or dying," as has been so emphatically stated by Novak,[4] that phase of the implantation theory is likewise just as dead. If endometrial tissue disseminated by menstruation is sometimes alive and capable of growing, if transferred to suitable situations, we might expect to find embolic lesions of this tissue in the

vessels of the uterine wall and even outside of that organ. If these lesions were found, they would furnish very strong evidence that they might have arisen from the implantation of endometrial emboli cast off by menstruation into the venous circulation.

I have recently reported[2] two such cases. Both patients were operated upon during their menstrual period. Bits of endometrial tissue were found in the venous sinuses of both uteri. An embolic or metastatic growth of endometrial tissue was found in a venous sinus of one uterus and many such lesions in the other uterus. Sufficiently complete serial sections showed that these growths either arose from or were implanted on the walls or linings of these vessels and did not arise from the invasion of the latter by endometrial tissue from without. These embolic-like

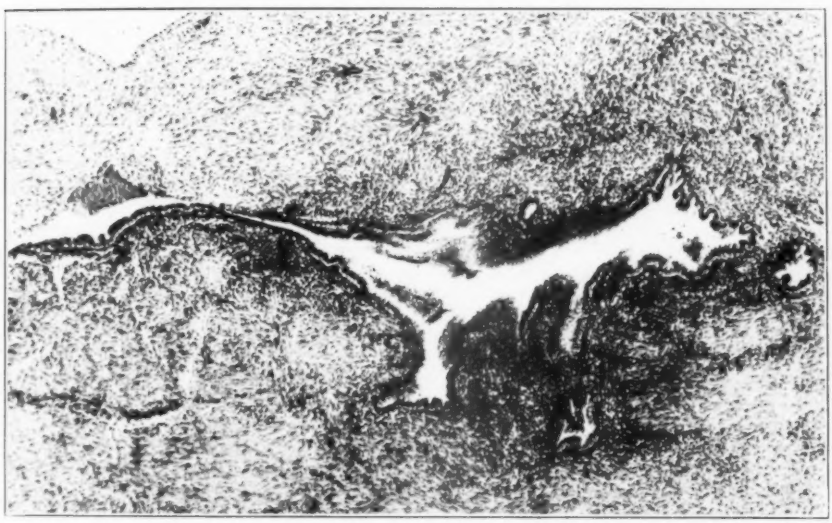


Fig. 9.—Photomicrograph (x 60) of an endometrial cavity of the posterior layer of the broad ligament adherent to the ovary. Blood is present in this cavity, and portions of its endometrial lining show the same reaction to menstruation (but not as marked) as that found in the mucosa lining the uterine cavity and the cyst of the ovary (Fig. 5).

growths of endometrial tissue must have originated either from a localized metaplasia of the endothelial lining of the veins and venous sinuses or else from the actual anchoring and implantation of bits of endometrial tissue similar to those found floating about in some of the vessels of the uterus. While an endometriosis of the direct type was present in a portion of the wall of each uterus, the distribution of the embolic lesions, as well as other histologic findings, indicated that the endometrial emboli, primarily responsible for the metastatic lesions, probably came from the mucosa lining the uterine cavity. Even if these emboli had been derived from the endometrial tissue of a direct endometriosis of the uterine wall, some traumatism would be necessary to rupture the endothelial lining of its veins and disseminate bits of endometrial tissue into their lumina. The reaction to menstruation is the most evident cause of such an injury.

Could bits of endometrial tissue escape into the venous circulation from the mucosa lining the uterine cavity by any other means than as a result of a menstrual reaction? We look to two other possible causes of this phenomenon, curettage and the termination of pregnancy by abortion or labor. Neither of the two patients, mentioned above, had ever been curetted and only one had been pregnant and that fourteen years before her operation.

Many interesting endometrial lesions were present in an endometriosis of the posterior vaginal wall of the second case. An endometrial cavity filled with menstrual blood and containing bits of endometrial tissue had

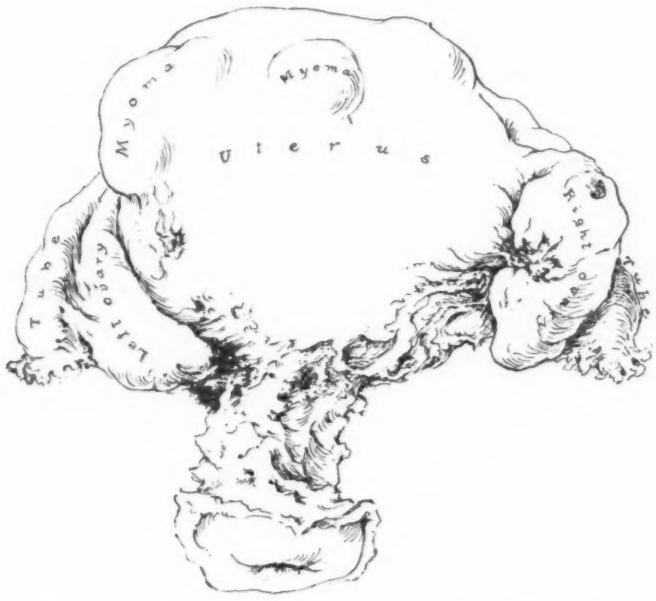


Fig. 10.—Posterior view of the uterus, tubes and ovaries (x $\frac{2}{3}$) removed for an extensive peritoneal endometriosis, fusing the posterior surface of the cervix with the sigmoid, thus obliterating the bottom of the culdesac. The uterus contains multiple leiomyomas; both tubes were *patent*. The right ovary is adherent by its *under surface* to the posterior surface of the uterus and the broad ligament of that side. The left ovary is adherent by its *lateral surface* to the posterior layer of the broad ligament. An attempt was made to remove the uterus, tubes, and ovaries without disturbing the attachment of the ovaries to their adjacent structures. This was only partially successful (see Figs. 11 and 13). The patient, fifty-three years old, was married but had never been pregnant.

almost eroded the overlying vaginal mucosa and was about to rupture and discharge its menstrual contents into the vagina (Fig. 1). From the study of this lesion, one could readily understand how a similar endometrial cavity in the ovary or any other pelvic structure might rupture and disseminate its menstrual contents into the peritoneal cavity. We could also understand that a similar endometrial lesion might rupture into an adjoining lymph vessel or capillary during its reaction to menstruation. The operation occurred on the second day of the men-

strual period. Bits of endometrial tissue were found lying free in the veins about other endometrial cavities of the vaginal wall and embolic growths of endometrial tissue were also present in these vessels. The actual escape of the menstrual contents of two of these cavities into a vein was seen. I can see only one correct interpretation of the etiology of the embolic endometrial lesions in the veins about these endometrial cavities and that is they arose from the implantation of endometrial tissue, disseminated into the veins from the menstrual rupture of the walls of the endometrial cavities into these vessels. If so, endometrial tissue disseminated by menstruation in this instance must have been alive and capable of growing, when transferred to suitable situations.

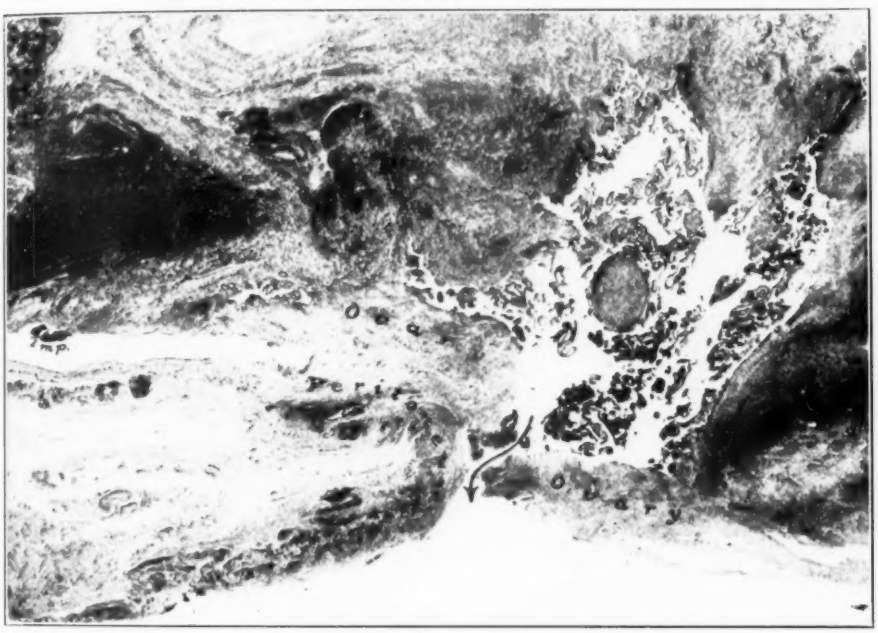


Fig. 11.—Photomicrograph (x 10) of a portion of the left ovary adherent by its lateral surface to the posterior layer of the broad ligament (Fig. 10). An endometrial cyst or cavity of the ovary, filled with fragments of endometrial tissue, is shown. Its wall is torn (see arrow) apparently due to the trauma of the operation and probably at the site of a previous perforation which had been subsequently closed by the fusion of the ovary with the posterior layer of the broad ligament at this point. An implantation-like patch of endometrial tissue (*imp.*) is situated on the lateral surface of the ovary and is similar to the fragments of endometrial tissue in the cavity and that filling the opening in its wall. This endometrial "implant" seems to be of more recent origin than the endometrial cavity, and, from the evidence presented in this section, might have arisen from tissue escaping through the perforation of the latter.

As a result of these studies in the menstrual dissemination of endometrial tissue into the venous circulation, the following three conclusions were drawn:

1. Fragments of endometrial tissue, at times, are disseminated into the venous circulation during menstruation from the mucosa lining the uterine cavity and also from ectopic endometrial foci.

2. Metastatic or embolic endometriosis may arise from the implantation of these emboli in near-by veins.

3. Endometrial tissue set free by menstruation, therefore, is sometimes not only alive but may actually grow, if transferred to situations favorable to its existence.

I might add a fourth conclusion. The veins and venous sinuses of the uterine and vaginal walls, under certain conditions, are favorable to the existence (growth) of endometrial tissue disseminated by menstruation.

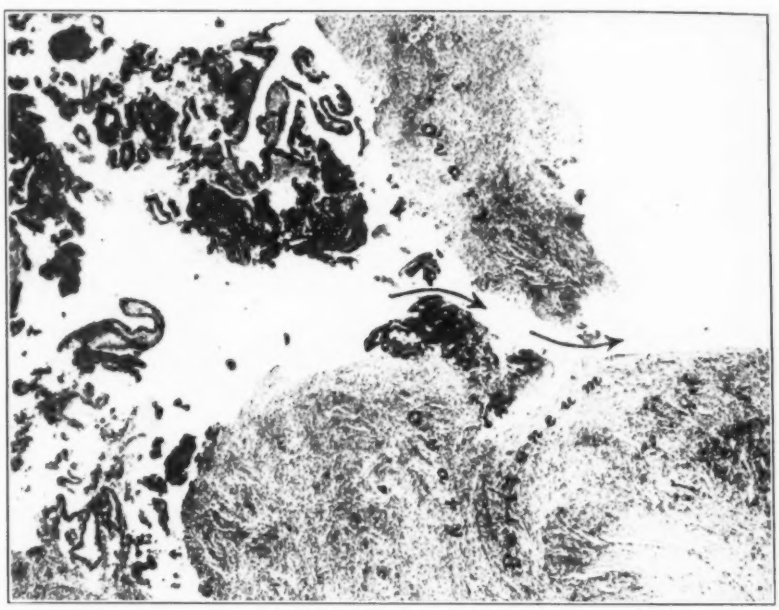


Fig. 12.—Photomicrograph (x 25) of the section, shown in the preceding illustration, at the site of the perforation of the endometrial cavity. The fusion of the fold of the peritoneum with the surface of the ovary indicates a reaction to some irritant escaping into the peritoneal cavity. The opening in the cyst wall indicates that this irritant may have been its contents. The endometrial "plug" filling the opening and attached to (implanted on) its sides suggests that this fragment of endometrial tissue had failed to escape through the perforation and was retained in this situation, or the endometrial lining of the cyst had grown out through the opening. Similar bits of endometrial tissue escaping from the cyst might have become implanted in other places, as the surface of the ovary (see *imp.* of the preceding illustration), the posterior surface of the uterus (Fig. 14), and even causing the peritoneal endometriosis, thus fusing the uterus with the sigmoid. The reaction of the endometrial lining of the cyst to menstruation would be the most likely cause of a perforation of the wall of the cyst.

EVIDENCE OF THE SUITABILITY OF THE PERITONEUM TO THE GROWTH OF ENDOMETRIAL TISSUE

If it can be shown that endometrial and tubal tissue will not grow on the peritoneum and cannot become implanted on it, no part of the implantation theory as an explanation of a cause of peritoneal endometriosis can be considered.

A study of the lesions of peritoneal endometriosis convinces one that endometrial tissue can and does grow on the pelvic peritoneum and also on the peritoneal surface of other structures which may be in the pelvic

cavity but primarily did not arise in it, such as the appendix, the cecum, loops of the small intestine, and their mesenteries. We must conclude that the visceral and parietal peritoneum is suited to the growth of endometrial tissue, whatever the origin of the latter may be.

Jacobson[5] has shown by his experimental work in rabbits and monkeys that bits of the uterine mucosa of these animals, when scattered in their peritoneal cavities, give rise to a peritoneal endometriosis similar to that found in human beings. It is natural to assume that the endometrial

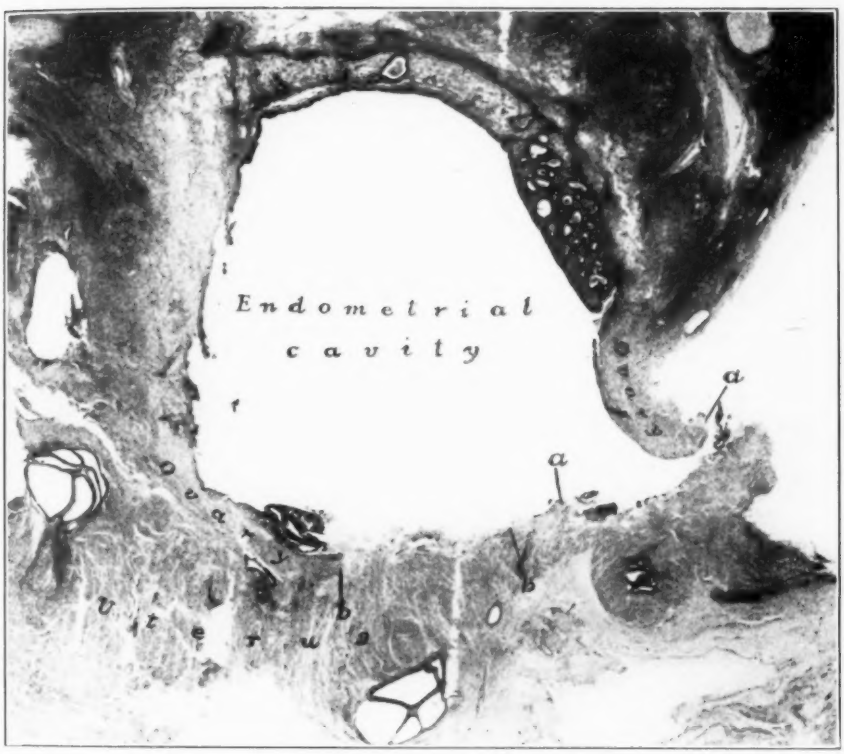
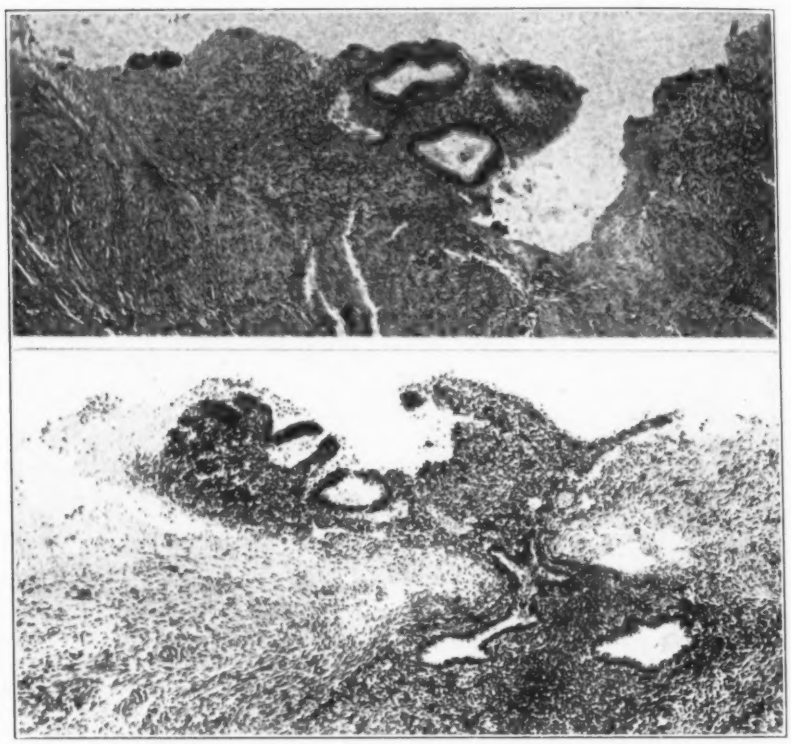


Fig. 13.—Photomicrograph (x 10) of a portion of the right ovary adherent to the posterior surface of the uterus (Fig. 10). An endometrial cyst or cavity of the ovary is shown with its wall *a*, torn from its former attachment to the uterus at *a* by the trauma of the operation. The chocolate-like contents of the hematoma or cyst escaped at that time. There is a gap *b-b* in the portion of the wall of the cyst adherent to the uterus. This gap is filled by the posterior surface of the uterus, thus completing or closing the endometrial cavity. This condition can be explained either by the rupture or by perforation of an endometrial cyst, and the subsequent closure of the opening by the ovary becoming adherent to the uterus or else endometrial tissue developing on the surface of the ovary caused adhesions (possibly due to its menstrual reaction) which fused the ovary to the uterus, and an endometrial cavity developed in this situation. In either instance menstrual blood might have escaped into the peritoneal cavity and a peritoneal endometriosis developed, as indicated by the "deposits" of endometrial tissue on the surface of and embedded in the uterine wall (see also Fig. 15).

tissue in these lesions arose from the implantation of the endometrial tissue "sown" on the surface of the peritoneum.

If bits of endometrial tissue, carried by menstrual blood escaping into the venous circulation of the uterus sometimes become implanted on the

endothelial surface of the veins and venous sinuses of the uterine wall and a like condition arises in the vaginal veins from the menstrual reaction of the mucosa of ectopic endometrial cavities in that situation, we might infer that bits of endometrial tissue, carried by menstrual blood escaping into the peritoneal cavity from any source, might become implanted on the mesothelial surface of the peritoneum and give rise to at least some of the lesions of peritoneal endometriosis.



Figs. 14 and 15.—Two photomicrographs (x 60) of implantation-like lesions on the posterior surface of the uterus. The first was situated beneath the distal pole of the adherent left ovary shown in Fig. 10, and would seem to be of more recent origin than the endometrial cavity of the ovary with evidence of perforation shown in Fig. 11. Similar lesions were also present on the lateral surface of the ovary. The second was situated near the right ovary (Figs. 10 and 13) also with evidence of rupture of its endometrial cavity and dissemination of its contents into the pelvis. If these near-by lesions arose from the escape of the contents of the endometrial cavities of the ovaries, the endometriosis of the culdesac might have had a similar origin.

The peritoneum of rabbits and monkeys evidently is adapted to the implantation of endometrial tissue disseminated in the peritoneal cavities of these animals, but is the peritoneum of women adapted to the implantation of this or any other tissue escaping into their peritoneal cavities?

Cancer escaping into the peritoneal cavity sometimes becomes implanted on the surface of the peritoneum, causing the lesions of peritoneal carcinosis. Could endometrial tissue escaping into the peritoneal cavity sometimes become implanted on the surface of the peritoneum

and cause the lesions of peritoneal endometriosis? Some will say "cancer is invasive and metastasizes by other channels." Endometrial tissue is sometimes also invasive and mestasizes by vascular channels.

We have shown that the peritoneum is suited to the growth of endometrial tissue, and it will be difficult to deny that endometrial tissue can become implanted on the peritoneum, if it can be demonstrated that this tissue escapes into the peritoneal cavity and the lesions of peritoneal endometriosis occur in situations and under conditions indicating their possible origin from such a source.

THE MENSTRUAL DISSEMINATION OF ENDOMETRIAL TISSUE INTO THE PERITONEAL CAVITY FROM ECTOPIC ENDOMETRIAL FOCI

Evidence has been presented of the viability of endometrial tissue disseminated by menstruation and that it will grow when transferred to suitable situations. We have shown that the peritoneum is suited to the growth of endometrial tissue and furthermore that cancer becomes implanted on it. We might infer that endometrial tissue which, at times, is invasive and metastasizes through vascular channels might also become implanted on the peritoneum. Should it be shown that endometrial tissue carried by menstrual blood escapes into the peritoneal cavity from any source, we would have strong presumptive evidence that under favorable conditions this tissue might become implanted on the peritoneum and cause peritoneal endometriosis. Should these lesions occur in situations and under conditions indicating or even suggesting their origin from such a source, this evidence would be still stronger.

Three possible sources from which menstrual blood might escape into the peritoneal cavity may be considered.

1. The menstrual rupture or perforation of the wall of an endometrial cavity (cyst), most frequently seen in the ovary.

2. The menstrual reaction of endometrial tissue growing on the peritoneal surface of the ovary or any pelvic organ or structure.

3. A back flow through the tubes from the uterus and also possibly from the menstrual reaction of the tubal mucosa.

We know that endometrial cavities in the vaginal wall, at times, rupture into the vaginal canal during menstruation and occasionally must discharge bits of endometrial tissue with the menstrual blood. (Fig. 1). Similar cavities in the abdominal scars of patients with endometriosis in that situation sometimes rupture and discharge blood at the menstrual period. We could infer that like cavities (cysts) in the ovary or in any pelvic organ or structure, at times, must rupture into the peritoneal cavity during menstruation. The study, at operation, of endometrial cysts or hematomas of the ovary shows that the ovary is usually adherent by its *lateral* or *under* surface to the side of the pelvis, posterior layer of the broad ligament, or the posterior surface of the uterus (Figs. 2,

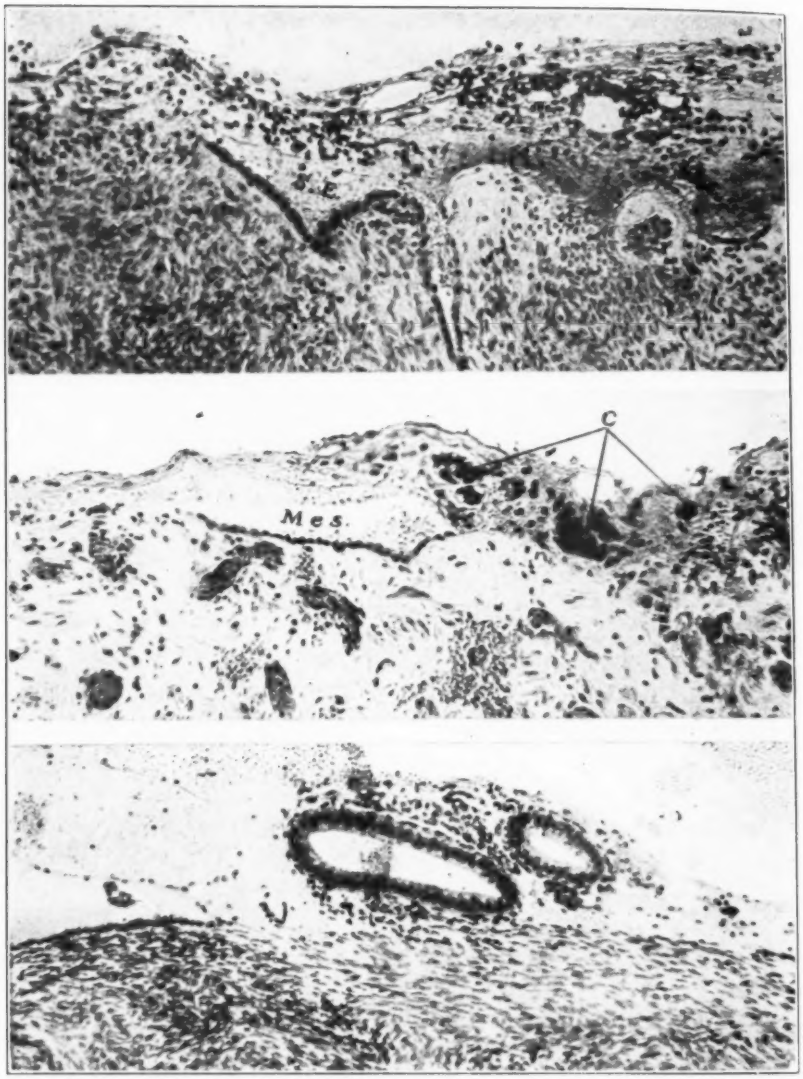


Fig. 16.—Photomicrograph (x 130) showing the reaction of the surface of the ovary to gonorrheal infection escaping through the tubes. An inflammatory exudate is present on the surface of that organ which consists of fibrin, leucocytes and wandering cells derived from the tissues of the ovary. The greater portion of the surface epithelium has disappeared. In the center of the photomicrograph it (*s.e.*) is still present and the exudate has arched over it. If the irritation had continued, the exudate would be replaced by granulation tissue and later by connective tissue with resulting thickening of the ovary or adhesion to adjacent structures. As the result of this reaction to infection, many interesting gland-like structures and cavities lined by the surface epithelium of the ovary arise from the inclusion of this epithelium and its subsequent growth, but they never develop into peritoneal carcinosis or true peritoneal endometriosis.

Fig. 17.—Photomicrograph (x 130) showing the reaction of the surface of the parietal peritoneum to the implantation of cancer, secondary to ovarian cancer. The exudate of Fig. 17, replaced by granulation tissue, is arched over the portion of the mesothelium (*mes.*) which has not disappeared, just as the exudate in Fig. 16 is arched over the surface epithelium of the ovary. Clumps of cancer cells (*c*) caught in the exudate now appear enmeshed in the granulation tissue. As a result of the peritoneal reactions in the implantation of cancer, many interesting lesions result.

Fig. 18.—Photomicrograph (x 130) showing the reaction of the surface of the ovary to the "implantation" of endometrial tissue. Multiple endometrial lesions were

10, 11 and 12). On freeing the ovary, the hematoma or cyst usually ruptures at the apparent seat of a previous perforation and its chocolate-like contents escape into the pelvis (Figs. 19 and 26). Adhesions are always present and often in situations easily soiled by material which might have escaped from a perforation of the ovarian cyst or hematoma. Endometrial tissue is often present in these adhesions and invades the structures involved by them. The question naturally arises: What is the relation between the endometrial tissue lining the cyst of the ovary and that scattered about in the implantation-like lesions of the associated peritoneal endometriosis?

The endometrial tissue in the ovary and that in the peritoneal lesions must have had a common origin or else one is secondary to the other. Should those involving the peritoneum arise from the escape of the con-

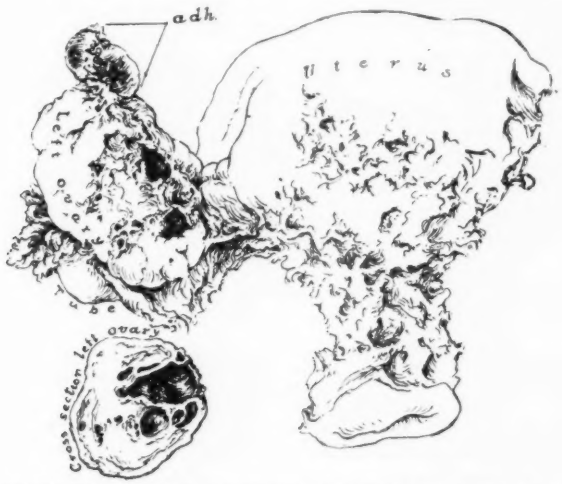


Fig. 19.—Posterior view of the uterus and left tube and ovary (x ⅔). The uterus was retroflexed and firmly adherent due to an extensive peritoneal endometriosis. The uterus, left tube, and ovary were removed and a portion of the right ovary was resected. An endometriosis superficially involved the greater portion of the posterior surface of the uterus. The left ovary was firmly adherent by its *lateral* and *under surfaces* to the posterior layer of the broad ligament and the posterior surface of the uterus. On freeing the ovary the characteristic chocolate-like contents of the ovarian hematoma escaped, possibly through the site of a previous perforation of the hematoma (see cross-section of the ovary). The ovary has been lifted up in order to show the adhesions and endometrial lesions on its *under* and *lateral surfaces*. Both tubes were *patent*. For photomicrographs of sections of the ovaries and uterus, see Figs. 20, 22, 23, and 24. The patient, thirty-six years old, had one child.

tents of the cyst, they must be due either to the stimulation of the peritoneum by some specific element of the cyst contents or else to the implantation of bits of endometrial tissue carried by the contents of the cyst escaping into the peritoneal cavity.

present on the *lateral surface* of the right ovary (see Fig. 29). The endometrial tissue in this illustration may have been derived either from the implantation of similar tissue from other endometrial lesions on the ovary (Fig. 30), from endometrial or tubal tissue escaping through the tubes (both were patent) or from a localized metaplasia of the surface epithelium of the ovary. The surface epithelium is intact at both ends of the illustration, but has disappeared beneath the endometrial tissue. The latter is enmeshed in an exudate somewhat similar to those shown in Figs. 16 and 17. Histologically it resembles an implantation lesion.

Clinical observations have taught us that these cysts sometimes rupture or perforate during menstruation and some of their contents escape into the pelvic cavity. A histologic study of ovarian endometrial cysts, removed during menstruation, demonstrates that the lining of these cysts sometimes reacts to menstruation, as does the mucosa lining the uterine cavity (Figs. 2 and 3), and bits of its endometrial tissue are disseminated into the cavity of the cyst. See Figs. 4 and 5. Therefore, they would be carried into the peritoneal cavity on rupture of the cyst. We have indications that endometrial tissue disseminated by menstruation is sometimes alive and will grow when transferred to suitable situations and also that the peritoneum is suited to the growth of endometrial tissue. Furthermore, the lesions of peritoneal endometriosis often occur in situations and under conditions indicating their origin from the escape of the contents of these cysts into the peritoneal cavity. The peritoneal reac-

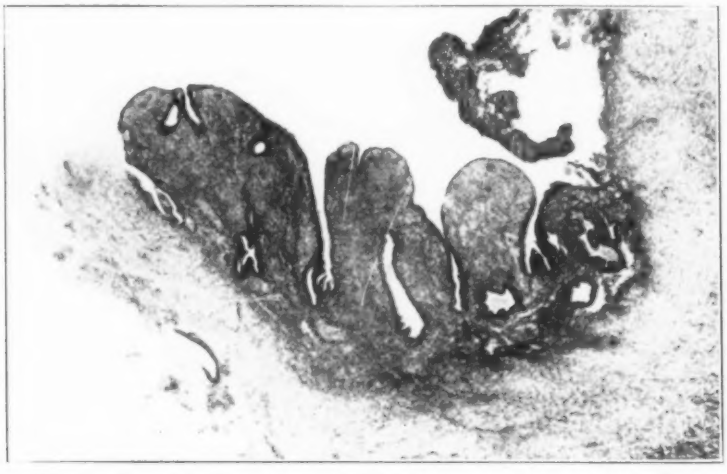
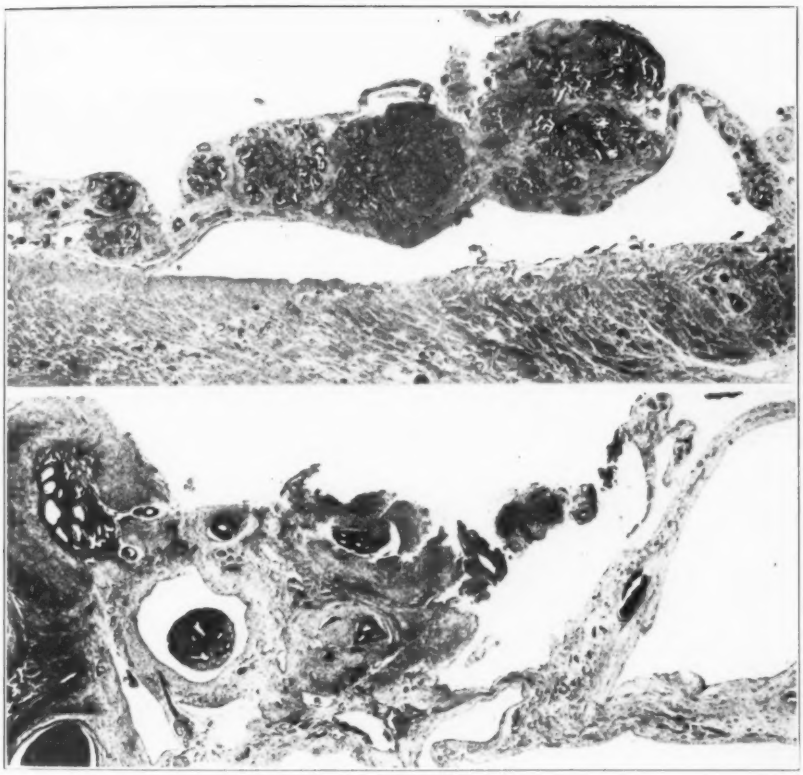


Fig. 20.—Photomicrograph (x 25) showing the lining of an endometrial cavity of the left ovary of Fig. 19 and near the apparent site of a previous perforation. Typical endometrial tissue is present.

tion to this tissue is similar to that of peritoneal carcinosis of implantation origin. We, therefore, have sufficient evidence to believe that peritoneal endometriosis sometimes arises from the implantation of endometrial tissue carried by menstrual blood escaping through a perforation of an endometrial (cavity) cyst of the ovary or any other pelvic structure. (Figs. 6, 10, 19, and 26.)

Endometrial tissue has been found growing on the surfaces of all of the pelvic organs and structures, including the cecum, appendix, loops of the small intestines, sigmoid, and their mesenteries. The patches of endometrial tissue on the ovary nearly always occur on the *lateral* and *under surfaces* of that organ, and these surfaces when they contain endometrial tissue are usually adherent to adjacent structures. The endo-

metrial tissue on the other organs and structures also is often surrounded by adhesions. Some of these patches of endometrial tissue, at times, evidently react to menstruation, just as does the uterine mucosa (Figs. 48, 49, and 50). Menstrual blood occasionally must escape from some of these endometrial foci into the peritoneal cavity. There are, therefore, strong indications that peritoneal endometriosis, at times, spreads in this manner. Because these foci are usually small and are often associated with adhesions and become embedded in the underlying organ



Figs. 21 and 22.—Two photomicrographs (x 10), the first of adhesions on the surface of the uterus, from a patient with peritoneal carcinosis secondary to ovarian cancer. It is but an exaggeration of the condition shown in Fig. 17. Bits of cancer had been caught in the exudate arising from the reaction of the peritoneum to cancer cells escaping into the peritoneal cavity. The subsequent transformation of the exudate into granulation tissue and then connective tissue, together with the growth of the cancer cells, evidently caused the lesion shown here. Compare with Figs. 22, 23, and 24.

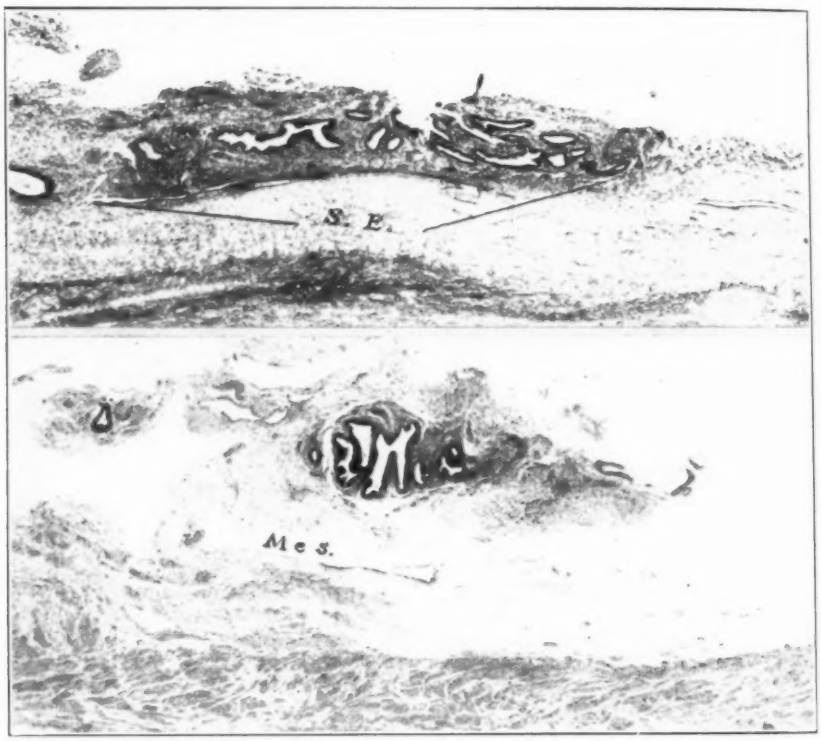
The second is of adhesions extending from the left ovary to the uterus, from a patient with peritoneal endometriosis associated with (or secondary to) ovarian endometriosis, with evidence that an endometrial cyst might have ruptured into the peritoneal cavity (Fig. 19). A portion of the ovary is shown at the left with endometrial tissue embedded in it. The origin of the endometrial tissue in these adhesions would seem to be similar to that of cancer in the adhesion of Fig. 21 and the latter was of implantation origin. See also Figs. 23 and 24.

or structure, the opportunity for the menstrual dissemination of endometrial tissue from them probably is not very great except early in their existence.

THE MENSTRUAL DISSEMINATION OF ENDOMETRIAL TISSUE THROUGH THE TUBES FROM THE UTERINE CAVITY

Objections have been made to the theory that bits of endometrial tissue are carried by menstrual blood escaping through the tubes and become implanted on the visceral and parietal peritoneum. The following are the more important of these objections:

1. Menstrual blood rarely, if ever, escapes from the uterine cavity into the tubes.



Figs. 23 and 24.—Two photomicrographs (x 25), the first of a patch of endometrial tissue on the *lateral surface* of the ovary (Fig. 19). An inclusion of the surface epithelium of the ovary is shown at *s.e.* The endometrial tissue is situated above it and evidently in ovarian tissue which has arched over the surface epithelium of the ovary as in the peritoneal reactions shown in Figs. 16, 17, and 21.

The second is of adhesions on the posterior surface of the uterus (Fig. 19), containing endometrial tissue. There has been a peritoneal reaction to some irritant causing adhesions and a mesothelial inclusion (*mes.*), the latter due to granulation tissue arching over the mesothelial surface of the peritoneum. Endometrial tissue is embedded in the adhesions above the mesothelial inclusion just as cancer of implantation origin becomes embedded in the adhesions of peritoneal carcinosis (Fig. 21). The peritoneal endometrial lesions possibly are of more recent origin than the endometrial cyst of the ovary and from evidence found at operation, it would seem that they might have arisen from the escape of the contents of the ovarian cyst into the pelvis (Fig. 19).

2. The lumen of the interstitial portion of the tube is too small for bits of endometrial tissue to pass through it.

3. Endometrial tissue, set free by menstruation, is dead or dying and therefore incapable of implantation.

4. Several days must be required for endometrial tissue to be carried from the uterine cavity through the tubes, and, therefore, there is little chance that "such degenerative tissue" after "probably many days of continuing degeneration and autolysis should grow where it falls." Novak.[4]

Novak[6] states that my theory would be greatly strengthened, if I could demonstrate two things.

1. The capacity of the degenerated endometrium given off at menstruation to grow in tissue culture.

2. The capacity of such endometrium to grow in the peritoneum or ovary of the human being or perhaps even of one of the lower animals.

I have examined menstrual blood obtained both from the vagina and also from the cavities of uteri removed at operation. The endometrial tissue obtained from these sources did not always show degenerative

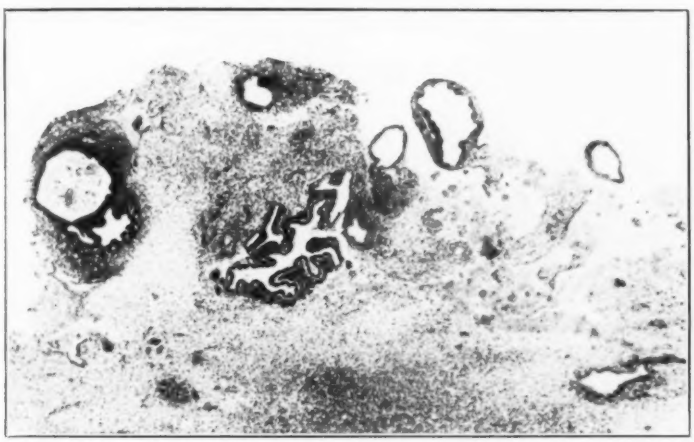


Fig. 25.—Photomicrograph (x 25) of a portion of the right ovary (case of Fig. 19). An apparent endometrial lesion was situated on its *lateral surface*. It is evidently of more recent origin than the endometrial cyst of the opposite ovary (see Fig. 19). Should the endometrial tissue in this case have arisen from the differentiation of celomic epithelium due to its stimulation by an ovarian hormone, we should expect that these lesions would all be of the same age.

changes, but frequently presented a normal appearance (Fig. 47). This, however, does not prove that it is alive and capable of growing, even if transferred to suitable situations. Evidence has already been given to show that endometrial tissue disseminated by menstruation into the venous circulation, at times, becomes implanted in the sinuses of the uterine wall and in one instance in veins about endometrial cavities in the posterior vaginal wall. Evidence has also been presented to show that endometrial cavities (cysts) of the ovary, at times, discharge their contents, containing endometrial tissue cast off by menstruation, into the pelvis and that peritoneal implantations of this tissue arise from this source. It has also been demonstrated by this and other observations that the visceral and parietal peritoneum is suited to the growth of endometrial tissue.

If bits of endometrial tissue could be carried through the interstitial portion of the tubes by menstrual blood escaping from the uterine cavity into these ducts and not spend too long a time in their transit, one would expect that peritoneal implantations of this tissue might occur from this source.

Over twelve years ago I began a series of experiments to determine the shape of the uterine cavity in normal and in pathologic conditions. The technic was as follows: the uterus removed at operation or autopsy was placed in a basin of warm water and then filled with melted gelatin (about 15 per cent) containing in suspension bismuth subcarbonate or barium sulphate. This was introduced through the cervical canal by means of a syringe. After filling the uterine cavity the syringe was withdrawn, the cervix clamped in order to prevent the escape of the injection mass and the specimen placed in cold water until the gelatin

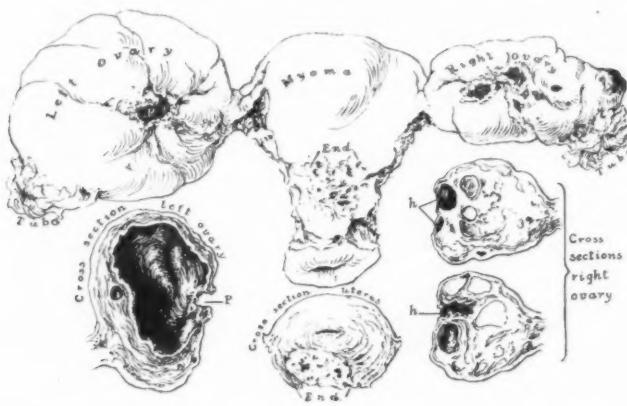
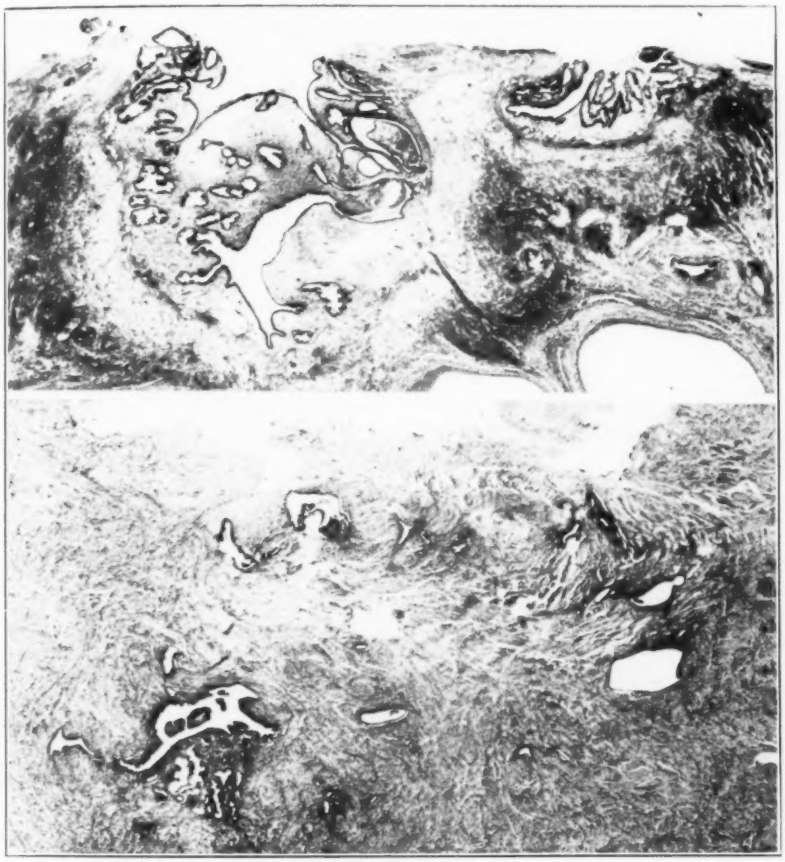


Fig. 26.—Uterus, tubes, and ovaries (x ½). The uterus was retroflexed and adherent due to peritoneal endometriosis. An intramural leiomyoma is present in the posterior wall of the body of the uterus. An endometriosis involved the uterus at the junction of the body and the cervix, invading its wall (see Fig. 28). The left ovary was adherent by its *lateral surface* to the side of the pelvis. On freeing it, the chocolate-like contents of the ovarian hematoma escaped, possibly through the site of a previous perforation of the hematoma (see also cross-section of the ovary). The right ovary was adherent by its *lateral surface* to the posterior layer of the broad ligament. Multiple endometrial lesions involved the posterior surface of the right ovary (see cross-sections of the ovary and Fig. 27). Both tubes were patent. The patient, thirty-one years old, was married but had never been pregnant.

had solidified. The uterus then was fixed in formalin. On filling the uterine cavity, the mass would usually easily escape through the tubes, if the latter were patent. Roentgenograms (especially stereoscopic ones) of the hardened uterus enabled one to obtain a clear picture of the form of the uterine cavity under various conditions and also of the course and relative diameters of the interstitial portions of the tubes. Many uteri were studied in this manner. In some the interstitial portion of the tubes appeared as a mere thread in the roentgenograms and in others as a relatively large canal. The results of some of these studies[7] were presented before this society nine years ago. Two of the illustrations of that paper are again reproduced, one showing a uterus with very

narrow interstitial portions of the tubes and the other with dilated ones (Figs. 40 and 41). I suggested at that time that menstrual blood under favorable conditions might escape through the tubes into the peritoneal cavity. Since then I have been greatly interested in the menstrual regurgitation of blood into the tubes and have operated upon many patients during their menstrual period and have occasionally observed this phenomenon (eight cases). I have also found endometrial tissue



Figs. 27 and 28.—Two photomicrographs (x 10), the first shows some of the endometrial lesions of the right ovary of the preceding illustration. They are evidently of more recent origin than the endometrial hematoma of the opposite ovary.

The second is of the endometriosis of the posterior uterine wall. There is apparently an invasion of the uterine wall from tissue implanted or developing on its peritoneal surface. If the former, it might have been derived from endometrial tissue escaping either from the ovary or from the tubes; both of the latter were patent.

in the lumina of tubes removed during menstruation. It seemed to me that the blood in the tubes in these eight cases came from the uterine cavity. It was present in both tubes in each instance. However, the source of endometrial tissue in the lumina of the tubes is a debatable one and if it can be shown that the interstitial portion of the tubes is

always so small that bits of endometrial tissue cannot pass through it, the peritoneal implantation of endometrial tissue from this source can be eliminated.

The roentgenograms of the injected uteri suggested that the interstitial portion of some tubes is sufficiently great to permit bits of endometrial tissue to pass through it (Figs. 40, 41, and 42). Can it be proved that blood escaping from the uterine cavity into the tubes will actually carry with it bits of endometrial tissue?

I have often observed blood dripping from the abdominal ostia of the tubes in abdominal operations which had been preceded by a curettage of the uterus. I do not understand just why this should occur. Bits of endometrial tissue, apparently set free by the curette, were

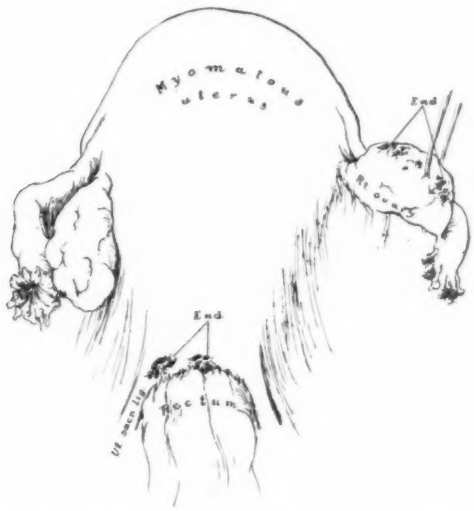


Fig. 29.—Uterus (containing a submucous leiomyoma), tubes, and ovaries drawn upwards (x ½), showing a peritoneal endometriosis obliterating the bottom of the culdesac and involving the left uterosacral ligament (Fig. 31). Multiple endometrial lesions are present on the *lateral surface* of the right ovary (Figs. 18 and 30). Both tubes were *patent*. The peritoneal endometriosis in the culdesac is in a situation easily soiled by material escaping from the tubes or ovaries, and that of the right ovary is on a surface of that organ, which would readily be contaminated by material escaping from the tube. The lateral surface of the ovary normally lies against the posterior layer of the broad ligament or side of the pelvis and thus a crevice is formed which would retain any material lodging in it. The lesions are situated on the dependent portion of the ovary, namely, the bottom of the crevice. The patient, forty-five years old, was single.

found in the lumina of tubes which had been removed and some of those pieces were larger than similar pieces of endometrial tissue set free by menstruation (Figs. 44 and 46). If endometrial tissue set free by curettage is carried by blood escaping from the uterine cavity into the tubes, similar tissue set free by menstruation might be carried with menstrual blood escaping from the uterine cavity into the tubes. Also, the time required for menstrual blood to go from the uterine cavity into the tubes might be very short, not several days but a few moments

and possibly no longer than that taken by uterine bleeding from a curettage.

These observations demonstrate that blood escaping from the uterine cavity into the tubes, at times, can carry with it bits of endometrial tissue and that the time required for this may be very short.

Fragments of endometrial tissue have been found in the lumina of tubes removed at operation. What is the possible origin of this tissue? I believe that endometrial tissue in the lumen of the tube may arise from the following sources; (1) artefacts; (2) carried by blood escaping from the uterine cavity during or after curettage; (3) from the menstrual reaction of endometrial tissue forming a part of the tubal mucosa; (4) from ectopic endometrial tissue in the pelvis (Novak); (5) carried by blood escaping into the tubes from the uterine cavity during menstruation.

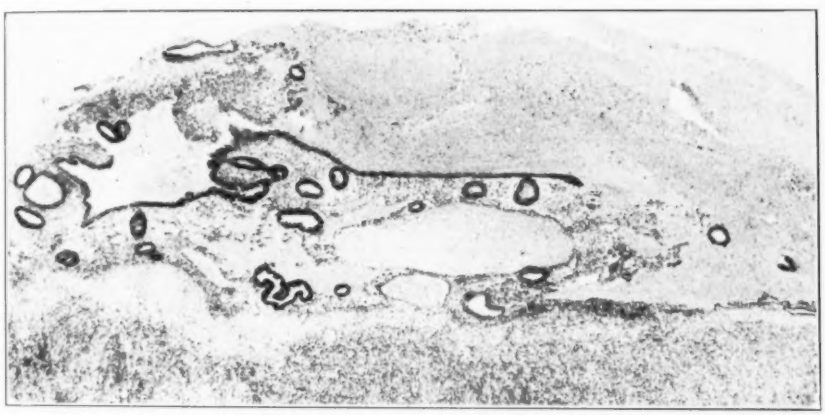


Fig. 30.—Photomicrograph (x 25) of a portion of the right ovary (Fig. 19). Typical endometrial tissue is present. In the menstrual reaction of this tissue bits of it might be set free, escape in the culdesac, or lodge in any of its natural pockets. They might also lodge on other portions of the ovary. Multiple endometrial lesions in different stages of development were present on the lateral surface of this ovary (see Fig. 19). They are often found on the peritoneal surface of structures adjacent to such an ovary. It is conceivable that such lesions on the surface of the ovary primarily might have arisen from endometrial tissue escaping through the tubes.

For some time I have been interested in the study of the menstrual dissemination of endometrial tissue into the venous circulation. Bits of the uterine mucosa were found in the veins and sinuses of menstruating uteri. A careful study of these sections and the technic of embedding the blocks, from which they were cut, convinced me that some of these findings were due to artefacts. The menstruating uterine mucosa is very friable; fragments break off during the embedding of the blocks of the uterus and some of these may lodge in the gaping veins and sinuses of the uterine wall. They appear as endometrial emboli in the stained sections (see Figs. 19, 20, and 21 of previous article[2]). If, however, fragments of the uterine mucosa are found

surrounded by blood in a vessel or attached to its wall by fibrin, it is evident that it reached this situation before the tissues were fixed. It occurred to me that bits of endometrial tissue floating about in the embedding solutions could lodge in the lumen of the ampulla of the tube as readily as in the gaping vessels of the uterine wall. A "block" of the ampulla of the tube would furnish a very convenient "basket" for any scraps of tissue floating about in the embedding solutions which were small enough to enter its lumen. Bits of uterine mucosa and barium sulphate were purposely placed in the solutions, in which blocks of tubes were being embedded. Both barium sulphate and pieces of uterine mucosa were found in sections from some of these tubes (Fig. 39). Just as faulty technic sometimes plays an important rôle in placing (disseminating) bits of endometrial tissue in the lumina of the gaping vessels of the uterine wall, it may also place such tissue in

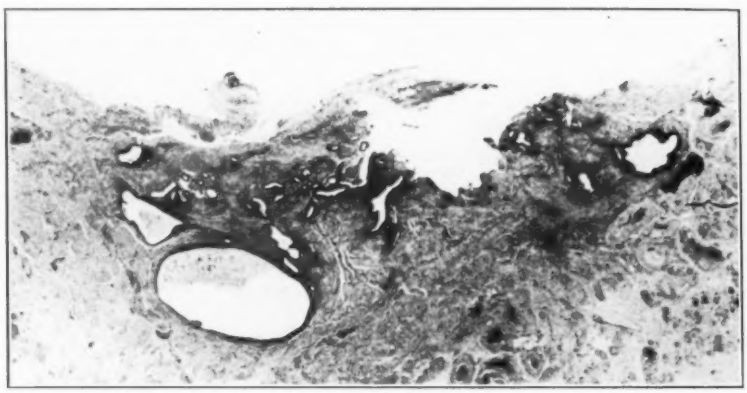


Fig. 31.—Photomicrograph (x 19) of the endometriosis of the left uterosacral ligament (Fig. 29). The endometrial tissue evidently has invaded this structure from its peritoneal surface. It was in a situation easily soiled by material escaping from the ovaries or the tubes. It must have arisen either by metaplasia of the peritoneum or from an implantation of endometrial tissue.

the lumen of the fallopian tube. It is difficult to estimate to what extent artefacts and curettage of the uterus have contributed to the etiology of the endometrial tissue in the tubes which have been described in the literature of this subject. This is a problem for each laboratory worker to decide for himself.

The histologic picture of tubes with bits of endometrial tissue set free by curettage differs from that of tubes containing endometrial tissue due to artefacts. In the latter the endometrial tissue lies free in the lumen of the tube (Fig. 39), while in the former it is embedded in blood (Fig. 46) which has obviously come from the uterine cavity, unless the blood has escaped from the tube after its removal. In recent years I have ligated the distal ends of tubes containing blood before removing them. An interesting problem arises and that is the fate of endometrial tissue carried by blood escaping into the tubes during

curettage. Could it sometimes be retained in the lumen of the tube and even live and actually increase in size or may it escape into the peritoneal cavity and sometimes become implanted on the pelvic peritoneum? Davis[8] has reported an interesting case suggesting the latter.

Theoretically it is possible for bits of endometrial tissue to be set free in the lumen of the tube from the reaction of the tubal mucosa to menstruation (Fig. 58).

Novak has published photomicrographs of sections of tubes showing endometrial tissue in their lumina and offers the theory that this tissue might have come from ectopic endometrial tissue in the pelvis and entered the tube through its abdominal ostium, just as the ovum and

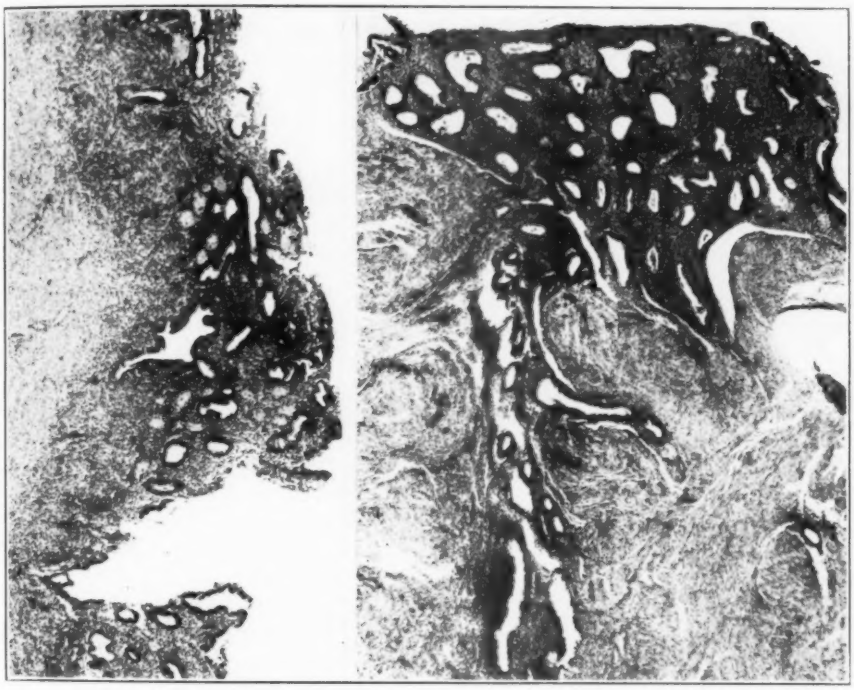


Fig. 32.—Two photomicrographs (x 25), the first of the lateral surface of the left ovary and the other of the adjacent peritoneum. The left ovary was adherent by its *lateral surface* to the side of the pelvis. Both *tubes* were *patent*. The peritoneal endometriosis was situated in the peritoneum adherent to the ovary. It would seem that the endometriosis of the ovary and of the peritoneum must have had either a common origin or one was secondary to the other. Both were in a situation readily soiled by material escaping from the patent left tube. The opposite tube and ovary were normal. Another peritoneal implantation-like lesion was present in the culdesac near the left uterosacral ligament (accidental findings in resection of the sigmoid for cancer in a patient, forty-nine years old, married, but who never had been pregnant).

particles of cancer in peritoneal carcinosis are known to enter the tubes. I believe that this occasionally might occur from the rupture of an endometrial cyst (cavity) and also from the menstrual reaction of endometrial tissue growing on the surface of the ovary and other pelvic structures. Endometrial tissue on the ovary nearly always oc-

curs on the *lateral* and *under surfaces* of that organ and the surface containing this tissue is usually adherent to adjacent structures, such as the side of the pelvis, the posterior layer of the broad ligament or the posterior surface of the uterus. On account of these adhesions it is difficult for bits of endometrial tissue to be set free even by a menstrual reaction so that they could escape into the tubes. However, I believe that it sometimes occurs and that they may escape into the lumen of the tubes.

During the last five years I have studied the material from 293 patients with peritoneal lesions containing endometrium-like tissue. In many of these cases one and sometimes both tubes and ovaries have been removed. In nearly all of the cases, in which one or both tubes have been removed, sections of the tubes have been examined. In some

Fig. 33.—View of the pelvic contents, uterus drawn forward (x ½). The fimbriae of the left tube were lightly adherent to the posterior layer of the broad ligament. In a shallow peritoneal pocket directly beneath the ostium of the tube, the endometrial lesion shown in Fig. 34 was obtained. A small nodule (end) is situated in the culdesac. Both ovaries appeared normal and both *tubes* were *patent*. Prior to the operation the uterus was retroflexed. The patient, thirty-four years old, was married and had children.

only one block was cut from the tube and only two or three sections from each block were studied. In other instances two or more blocks from the tube or tubes were made and many sections were studied from each block. The purpose of these studies was threefold: (1) to detect fragments of endometrial tissue in the lumen of the tube; (2) to seek evidence of a menstrual reaction of the tubal mucosa; (3) to compare the histologic structure of the tubal mucosa with that of the associated ectopic endometrial tissue. I have encountered only three instances (Figs. 46, 53, and 55) of fragments of endometrial tissue in the lumen of tubes from these cases and all three patients

were menstruating at the time of the operation. Blood was observed in both tubes in each instance at the time of the operation and it was apparently coming from the uterus. At the time I thought (and still believe) that the pieces of endometrial tissue in the tubes were probably carried by the blood coming from the uterine cavity, as blood escaping from the uterine cavity during curettage sometimes carries with it endometrial tissue.

Let us consider the possible sources of blood in the lumina of the tubes of patients operated upon during menstruation: (1) the tubal mucosa; (2) the uterine cavity as the result of curettage; (3) drawn into the tube through its abdominal ostium from blood in the pelvis; (4) as a back flow from blood in the uterine cavity.

Bleeding occasionally occurs during menstruation from ectopic patches of uterine mucosa in the tubal lining (Fig. 58). Should menstruating

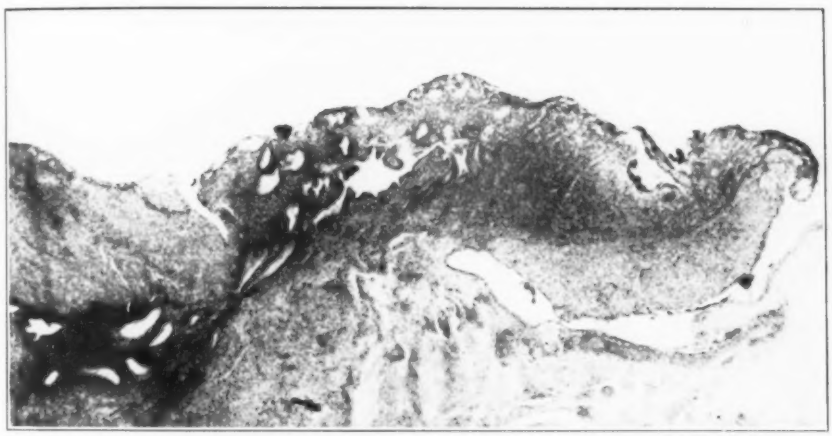


Fig. 34.—Photomicrograph (x 25) of the endometrial lesion obtained from the peritoneal pocket beneath the ostium of the adherent left tube of Fig. 33. Endometrial tissue implanted or developing on the surface of the peritoneum has invaded its deeper structures. It would seem to have arisen from material escaping from the tube. Is it an implantation of endometrial tissue or a metaplasia of the peritoneum arising from stimulation of the latter by some specific material escaping from the tube?

uteri be curetted prior to abdominal operations, one would expect to find blood in the lumina of the tubes in some of these cases, just as it occurs in the tubes of nonmenstruating uteri following curettage.

One would also expect that blood in the pelvic cavity from any source might be drawn into the tube through its abdominal ostium. This last year I operated upon a patient, apparently during her menstrual period. There was a large amount of blood in the pelvis and it could be seen dripping from the right tube which was only slightly larger than the opposite one. The left tube appeared normal and on stripping it blood did not escape from its ostium. The bleeding tube was removed and was found to contain the remains of a very early pregnancy. I have since examined the opposite patent tube in two

other cases of tubal pregnancy and was unable to detect any blood in them. I have found it in the opposite tube when the fimbriated end has been occluded. These observations are too few to be of any great value.

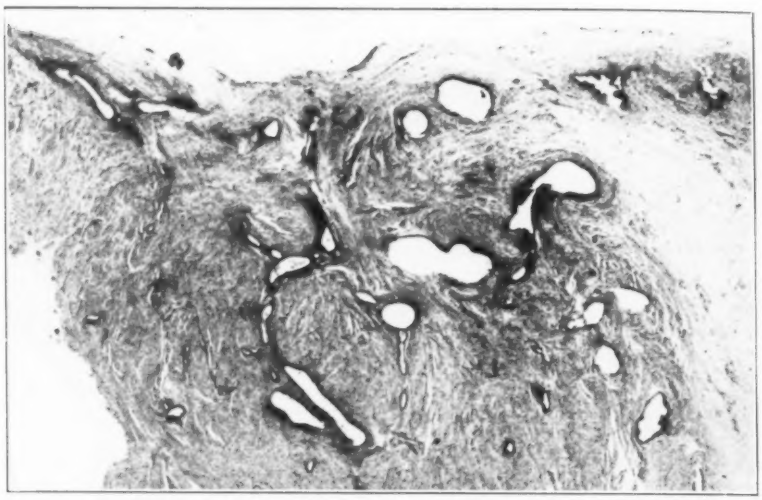


Fig. 35.—Photomicrograph (x 10) of the nodule removed from the culdesac of Fig. 33. It is apparently an older lesion than that shown in Fig. 34. It must have arisen from an earlier implantation of endometrial tissue or an earlier specific stimulation of the peritoneal mesothelium.

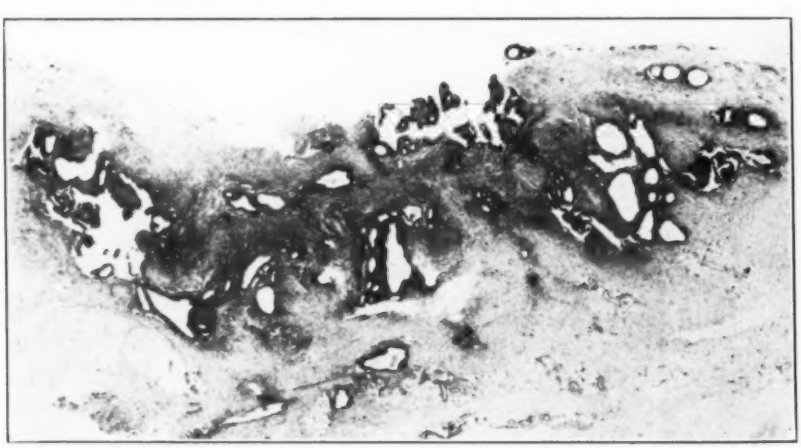


Fig. 36.—Photomicrograph (x 10) of a "patch" of endometriosis in the peritoneum of the side of the pelvis, very close to the ostium of the patent left tube and directly over the ureter, as was the peritoneal endometriosis of Fig. 32. A similar lesion involved the left uterosacral ligament. Both ovaries were normal and both tubes were patent. The patient, forty-two years old, had children. It is an implantation-like lesion in a situation easily soiled by material escaping from the patent left tube.

When blood is found at operation in the tubes of menstruating uteri and apparently is increased by compressing the uterus, this blood probably came from the uterine cavity and, at times, must carry with it bits of endometrial tissue set free by menstruation.

In four instances in which blood was observed by me, escaping from the tubes of patients operated upon during menstruation, there was no indication for the removal of the tubes other than the desire to study their contents. Blood was collected from the tubes by stripping them from the uterus towards their fimbriated end. The blood was spread on slides, dried, fixed, and stained by various methods. Epithelium-like cells were found in these preparations and also clumps of cells resembling the stroma of the uterine mucosa. There are certain valid

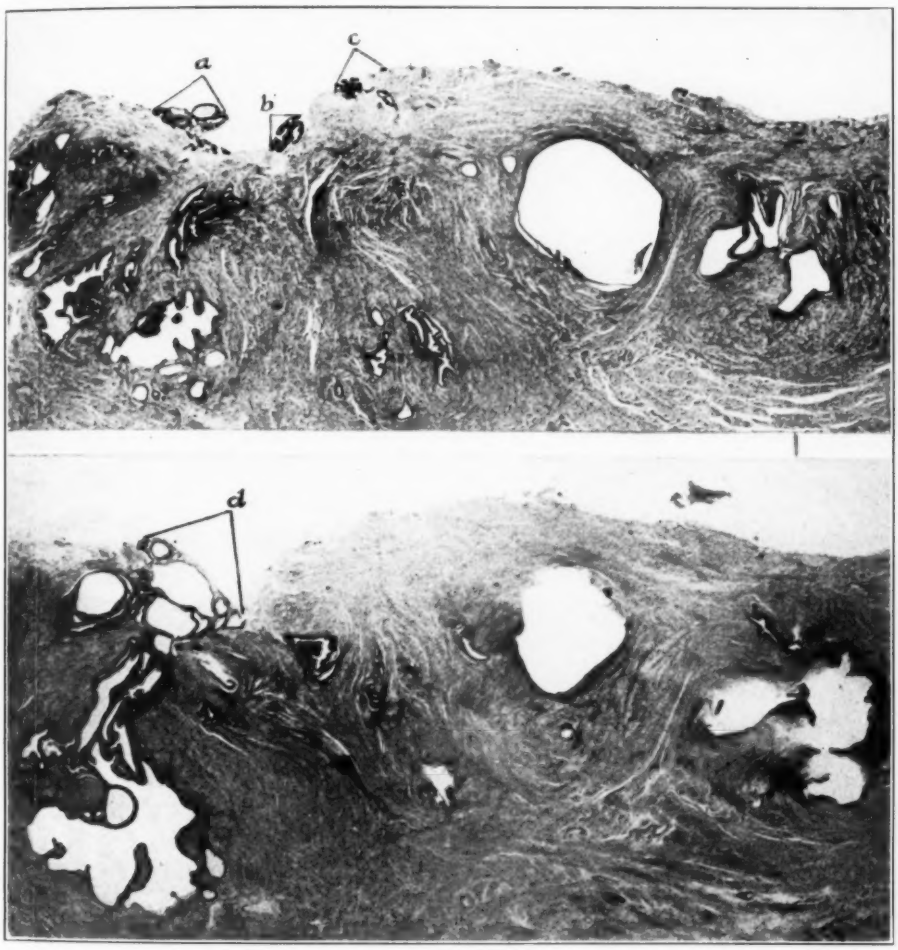


Fig. 37.—Two photomicrographs (x 10) of sections of an endometriosis of the posterior uterine wall at different levels of the same block. Typical endometrial tissue is growing on the peritoneal surface of the uterus at *a* and *b*. Lesion *a* is a part of the endometrial tissue of *d* on the peritoneal surface of the uterus shown in the lower photomicrograph. The latter apparently has invaded the deeper tissues of the uterine wall, causing a so-called adenomyoma. For a higher magnification of the lesion indicated by *c* of the upper photomicrograph, see next illustration. The patient, fifty years old, was single. The uterus was retroflexed and contained multiple leiomyomas. Both *tubes* were *patent* and both ovaries appeared normal. The left ovary was removed and endometrial tissue was not found in it. Implantation-like patches of peritoneal endometriosis were present on the posterior surface of the lower portion of the body of the uterus and in the posterior culdesac in situations easily soiled by material escaping from the patent tubes.

objections to this method. One is that trauma of stripping the tubes may dislodge some of the tubal mucosa and the other is the difficulty we (Dr. L. A. Sutton who was interested in this problem and myself) experienced in identifying the cells in these specimens. In all four cases blood was present in both tubes and in none of them was a peritoneal endometriosis present or any evident endometrial tissue on the surface of the ovaries.

In four other cases one or both tubes were removed with the uterus.

CASE I.—Peritoneal endometriosis with an adherent, retroflexed uterus. Endometrial lesions were present on the lateral surface of the ovary, the posterior surface of the uterus, and on the cecum. Blood was observed escaping from both tubes on exposing the pelvic organs at the operation. The uterus, right tube and ovary

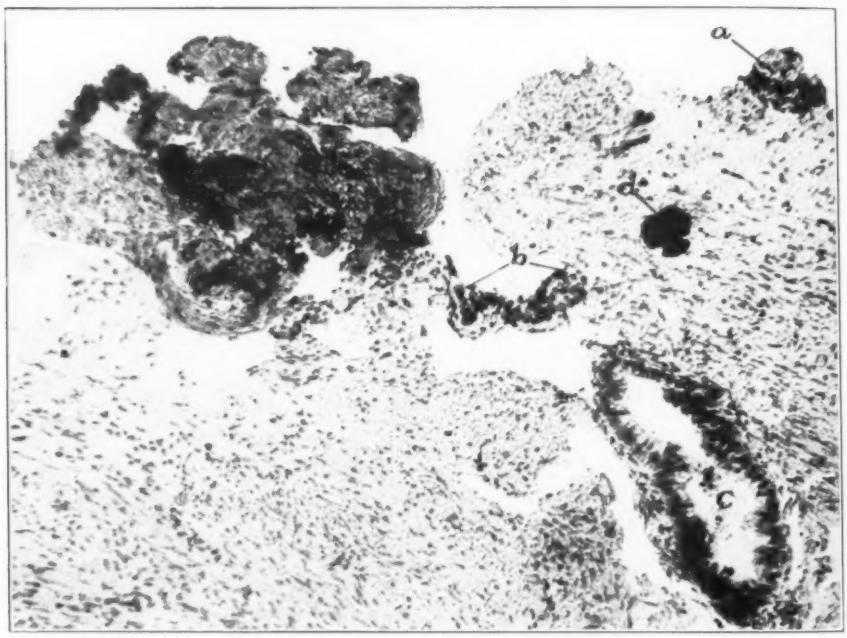


Fig. 38.—Photomicrograph (x 130) of the peritoneal lesion indicated by *c* of the preceding illustration. The surface of the uterus shows a reaction to some irritant. At *a* a piece of tissue consisting of fibrin and "dead cells" is adherent to the uterus. A larger mass of similar tissue is present to the left. At *b* endometrium-like cells have grown over or are attached to a "bridge" of newly-formed tissue. Possibly these cells were derived from similar cells in the necrotic mass above them and to the left, which had not died. Endometrial tissue *c* is becoming embedded in the uterine wall by the tissue of the latter growing over it. Lesion *d* probably represents a mass of cells which are dead. This and other sections from the same block suggest a peritoneal reaction to endometrial tissue added to (implanted on) the posterior surface of the uterus. The greater portion of this tissue in this photomicrograph is dead, but some lived (that in contact with the surface of the uterus) with a resulting endometriosis.

were removed. Unfortunately, the distal end of the tube was not ligated and most of the blood in the tube escaped during the manipulation of the operation. Epithelium and tissue resembling endometrial stroma were found in the lumen of the tube, adherent to the surface of the tubal mucosa. From the nature of

the peritoneal lesions and those in the ovary I do not believe that endometrial tissue in the tube could have come from these (Figs. 51, 52, and 53).

Case 2.—Retroflexed uterus containing multiple small leiomyomas. On exposing the pelvic organs at operation blood was observed escaping from both tubes and apparently coming from the uterus. Two small hemorrhagic blebs were present on the under surface of the left ovary. Blood was not observed escaping from these. The fimbriated ends of both tubes were first ligated and also the uterine end of the right tube, then the uterus, left tube and ovary, and right tube were removed. This case was reported in a previous article.[9] Strips of epithelium, a gland, and fragments of endometrial stroma mixed with blood were found in the lumen of one of the tubes. These were identical in their structure and staining reaction with those present in the mucosa of the uterus from which a large amount of tissue had been cast off by menstruation. The ovarian lesion consisted of two

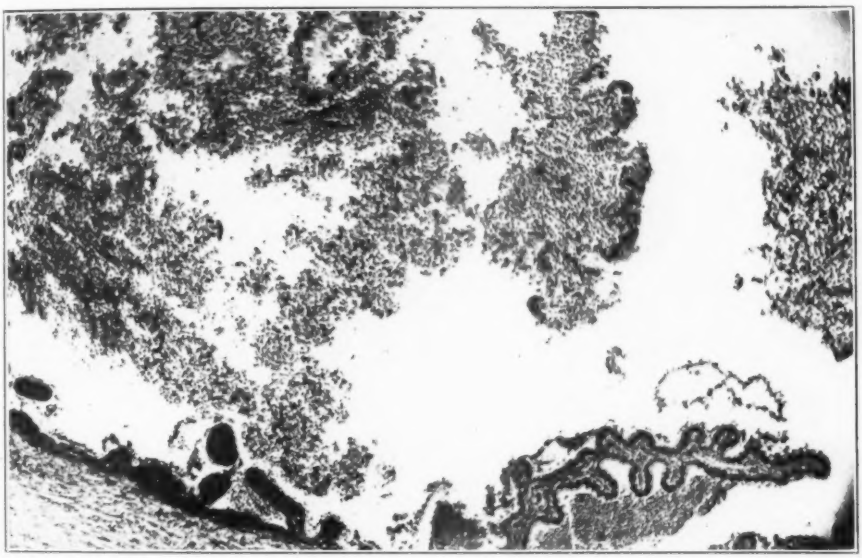


Fig. 39.—Photomicrograph (x 60) showing endometrial tissue free in the lumen of the tube due to an "artefact." Since bits of endometrial tissue floating about in the embedding solutions easily lodge in the empty vessels of blocks of the uterine wall carried through these solutions (see Figs. 19, 20 and 21 of previous article[2]), similar scraps in these solutions might find a "block" of the ampulla of the tube a very convenient "basket" in which to drop. Bits of endometrial tissue and barium sulphate were purposely placed in the embedding solutions through which "blocks" of the tubes were being carried and these were found in sections of the tubes. In a study of endometrial or any other foreign tissue in the lumen of the tube an artefact should be considered, and, if possible, eliminated.

small blebs lined by epithelium of endometrial type with very little stroma and no glands. In places a small amount of the epithelial lining had been cast off by an apparent menstrual reaction or from the trauma of the operation, but not enough to account for that present in the tube nor did it resemble this as closely as did the mucosa lining the uterine cavity. The blood in the tubes was apparently coming from the uterus and carried with it endometrial tissue. It might be claimed that the endometrial tissue was forced into the tubes by the manipulation of the uterus during the operation, but blood was in the tubes prior to the handling of the uterus (Figs. 54, 55, and 56).

Fig. 40.—Roentgenogram (x ⅔) of the uterus, tubes, and ovaries after filling the uterine cavity with gelatine containing bismuth subcarbonate. The mass readily escaped from the uterine cavity into the tubes. The uterus, tubes, and ovaries were removed for uterine bleeding due to myofibrosis (illustration published[2] in 1918). The interstitial portions of the tubes appear as mere threads, but even so, small bits of endometrial tissue might be carried into the tubes by blood escaping from the uterine cavity during curettage and menstruation (see Fig. 42). The specimen was hardened in formalin, before taking the roentgenogram, which would narrow the lumen of the tube. The uterus was placed, anterior surface downward, on a piece of waxed cardboard, beneath which was the photographic plate in two light-proof envelopes. Therefore, the distance from the tubes to the photographic plate was short. This would cause only a slight enlargement of the apparent lumen of the tube, possibly not compensating for the shrinkage due to hardening the specimen.

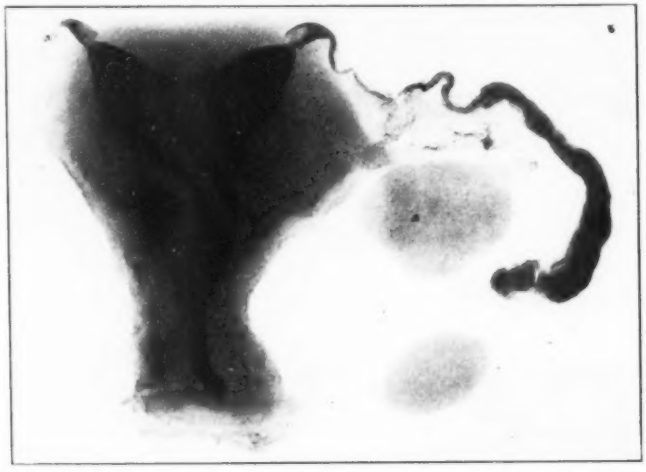


Fig. 41.—Roentgenogram (x ⅔) similar to the preceding. The uterus and one tube and ovary were removed for multiple small uterine leiomyomas. The lumen of the interstitial portion of the tube is much greater than that of the preceding specimen, and therefore, larger bits of endometrial tissue could pass from the uterine cavity into the tubes than in the former. Illustration also published in 1918, in the legend of which I suggested that menstrual blood, at times, might escape through the tubes into the peritoneal cavity. Note the normal construction of the distal end of the tube at the origin of its fimbriae, also shown in the preceding specimen.

CASE 3.—Leiomyoma of the uterus. On exposing the pelvic organs at operation blood was observed escaping from both tubes. Endometrial tissue was present on the lateral surface of the left ovary which was adherent to the posterior surface of the uterus. A small patch of endometrial tissue was present on the posterior surface of the uterus mesial to the left ovary. The distal ends of both tubes were ligated and then the uterus, both tubes, and ovaries were removed. Bits of endometrial tissue were found embedded in blood in the lumen of the tubes, which were identical in their structure with fragments of the uterine mucosa present in blood obtained from the uterine cavity. The endometrial tissue on the sur-

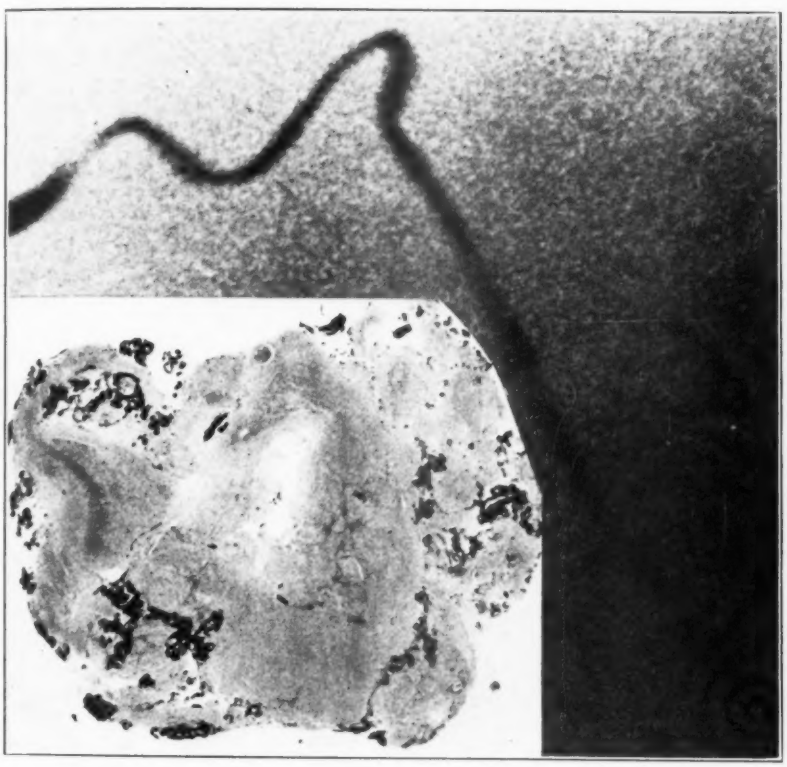


Fig. 42.—Photomicrograph (x 10) of a section of menstrual blood from the cavity of the uterus shown in the next illustration, placed beside an enlargement (x 10) of the very narrow thread-like interstitial portion of the tube shown in the roentgenogram of Fig. 40. The smaller bits of the uterine mucosa in this blood would easily pass through the lumen of the tube and the larger pieces would readily pass through a tube of greater caliber as the tube of the roentgenogram of Fig. 41. Blood escaping from the uterine cavity into the tubes during curettage and menstruation, at times, might carry with it bits of the uterine mucosa suspended in that blood (Fig. 46).

faces of the ovary and the uterus showed the same reaction to menstruation as did the mucosa lining the uterine cavity. I cannot exclude the possibility that blood, carrying with it endometrial tissue, had escaped into the abdominal ostia of the tubes from the menstruating endometrial tissue on the ovary and the uterus. From observations made at operation it appeared that the blood in the tube came from the uterine cavity. It was present in both tubes. The endometrial lesions on the surface of the left ovary were small and that aspect of the ovary was adherent to the uterus, thus lessening the chance of endometrial tissue escaping (see Figs. 43, 45, 46, 48, 49, and 50).

CASE 4.—Submucous leiomyoma of the uterus. On exposing the pelvic organs at operation blood was observed escaping from both tubes and apparently coming from the uterus. Peritoneal endometriosis was not present, nor was there any evidence of endometrial tissue on the surface of the ovaries. The fimbriated end of the left tube was ligated and the left tube and ovary and uterus were removed. A clump of epithelium-like cells, which I was unable to identify, was found in the lumen of the tube but no endometrial stroma. A mitotic figure was present in one of these cells (Fig. 57). I was unable to find any mitotic figures in epithelial cells in the blood obtained from the uterine cavity.

I fully realize that it is impossible definitely to state the origin of the blood in the lumen of the tubes in these eight cases. In all instances the blood was observed in the tubes of menstruating uteri prior to the opera-

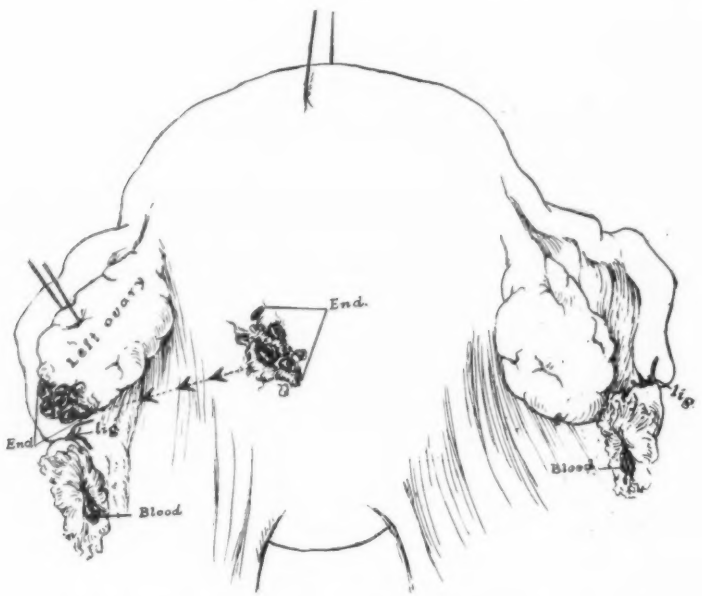


Fig. 43.—Myomatous uterus, tubes and ovaries drawn upwards (x ⅔). (Case 3 of this article.) The patient, forty-nine years old, married and having had children, was operated upon during menstruation. The left ovary (freed and lifted up) was adherent by its *lateral* and *under surfaces* to the posterior uterine wall, due to an endometriosis of the ovary (Fig. 48). A patch of endometrial tissue (Fig. 49) is present on the posterior uterine wall adjacent to the area to which the ovary had been adherent. Blood was observed coming from the abdominal ostia of both tubes. The distal ends of both tubes were first ligated in order to retain their contents, and then the uterus, tubes, and ovaries were removed. Did the blood in the tubes come from the uterine cavity or were the tubes disgorging blood obtained from the menstrual reaction of the misplaced endometrial tissue on the surface of the uterus and the lateral and under surface of the left ovary, the latter having been adherent to the uterus? Blood was present in *both tubes* and apparently was coming from the uterus and I believe that was its origin.

tive manipulation of that organ and if it came from the uterine cavity, as it apparently did, we know that it may carry with it bits of endometrial tissue. We should also expect that this endometrial tissue would be as capable of becoming implanted in suitable situations as that escaping into the venous circulation of the uterus and vagina and that escaping into the peritoneal cavity from ectopic endometrial foci in the pelvis.

THE LESIONS OF PERITONEAL ENDOMETRIOSIS OFTEN OCCUR IN SITUATIONS AND UNDER CONDITIONS INDICATING THEIR ORIGIN FROM MATERIAL ESCAPING INTO THE PERITONEAL CAVITY

One of the outstanding features of patients with peritoneal endometriosis is that the tubes are usually patent. In 293 cases of peritoneal lesions containing endometrium-like tissue encountered during the last five years both tubes appeared to be patent in 284, a unilateral hematosalpinx in three (in two of these blood was present in the opposite tube but the tube was patent), bilateral hematosalpinx in four, and bilateral pyosalpinx in two. Patent tubes apparently increase the incidence of peritoneal endometriosis and possibly the relatively large number of

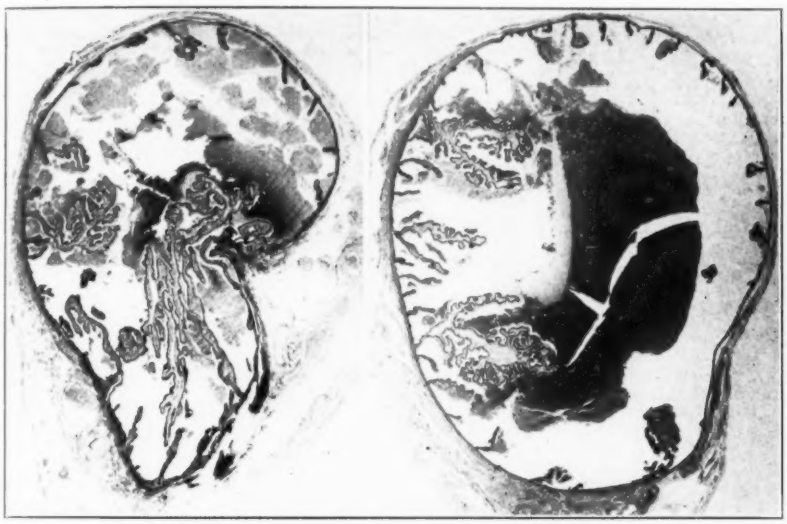


Fig. 44.—Photomicrograph (x 6) of a cross-section of the ampulla of a tube containing blood and bits of endometrial tissue (Fig. 46), carried with that blood from the uterine cavity during curettage. The abdominal operation had been preceded by a curettage of the uterus and repair of the pelvic floor. On exposing the pelvic contents, blood was observed dripping from the abdominal ostia of both tubes. I had planned to sterilize the patient. One tube was removed after ligating its distal end, so as to retain its contents. This tube was fixed in formalin. The opposite tube was cut at its uterine end and the stump buried. The bits of endometrial tissue (Fig. 46) floating about in blood in the lumen of the tube must have passed through its interstitial portion. Therefore, similar bits of endometrial tissue might be carried into the tube by menstrual blood escaping from the uterine cavity (Fig. 46).

Fig. 45.—Photomicrograph (x 6) of a cross-section of the ampulla of one of the tubes shown in Fig. 43. It contains blood and small bits of endometrial tissue, possibly carried with blood from the uterine cavity during menstruation, just as blood escapes from the uterine cavity into the tubes during curettage (Figs. 44 and 46). The ligated tubes, hardened in formalin, were cut into blocks and many sections were made from each block. Bits of endometrial tissue were found in sections from two different blocks. Unfortunately, the blocks removed from both tubes were run through the embedding solutions together and I was unable to determine whether the endometrial tissue was found only in one tube or in both. Blood was present, however, in both tubes.

patients with hematosalpinx (seven) may be of some significance. In the six cases with occlusion of both tubes, the peritoneal lesions might have been present prior to the closure of the fimbriated ends of the

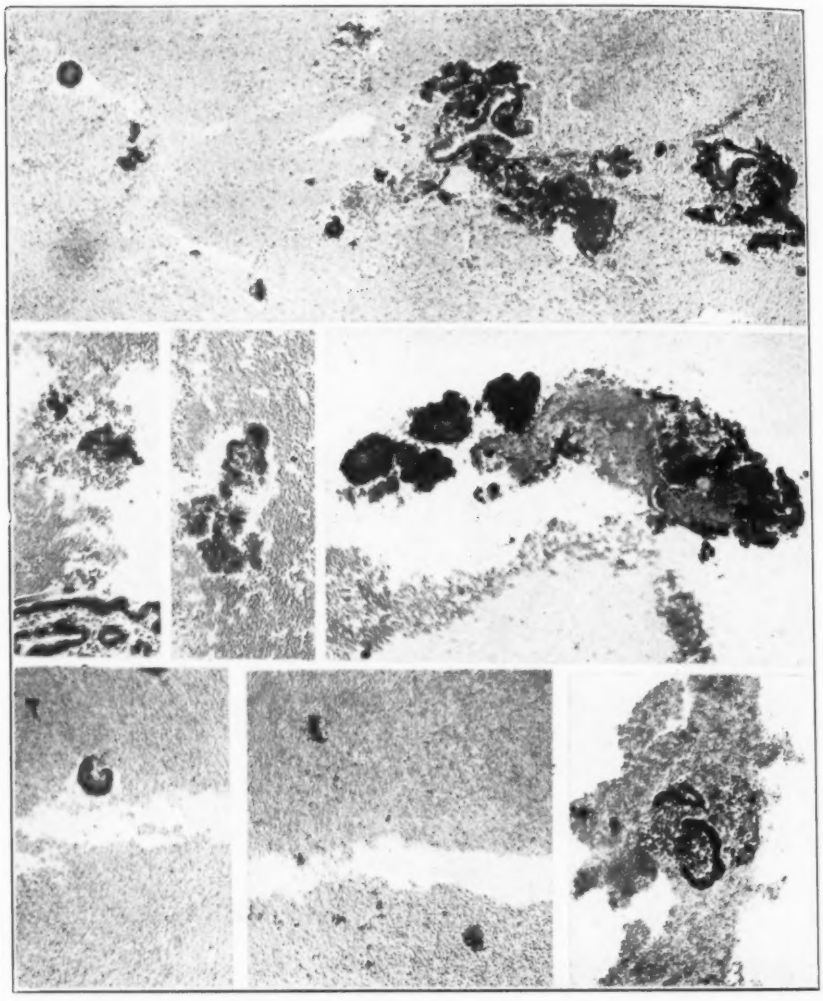


Fig. 46.—Seven photomicrographs (x 60), the lower three are from sections of the tube shown in Fig. 44 containing blood which had escaped from the uterine cavity during curettage. Bits of uterine mucosa are present in the blood and must have been carried with the latter through the interstitial portion of the tube.

The upper photomicrograph is of the section of the blood from the cavity of the menstruating uterus shown in Fig. 43. Blood apparently coming from the uterus was present in both tubes. Did the blood and endometrial tissue in these tubes come from the uterine cavity or from the menstruating ectopic endometrial tissue on the surface of the left ovary and adjacent surface of the uterus (Fig. 43, 48, 49, and 50)?

The middle three photomicrographs are from three different sections of the tube or tubes shown in Figs. 43 and 45. The first two photomicrographs show bits of endometrial tissue (epithelium and stroma) similar to those in the photomicrograph of the blood from the uterine cavity (above) and also those carried into the tubes during curettage (below). This suggests that they came from the uterine cavity but does not prove it. Both blood and endometrial tissue might escape into the tubes from the menstruating endometrial tissue on the surface of the ovary and the uterus (see Figs. 48, 49, and 50). The largest of the three middle photomicrographs shows a curved, elongated, "moulded" mass consisiting of bits of endometrial tissue and fibrin. Should the endometrial tissue in the photomicrogrpah above it be forced into a narrow, curved tube, it would be moulded or fashioned into a mass like this. I believe that this moulded mass represents a cast of the lumen of a narrow portion of the tube and adds to the evidence already presented that the endometrial tissue in the tube or tubes was derived from the uterine cavity. The manipulation of the uterus during the operation may have forced endometrial tissue and blood from the uterine cavity into the tubes and dislodged this endometrial "plug." This is probably true. Blood was, however, observed escaping from the tubes before the uterus was handled.

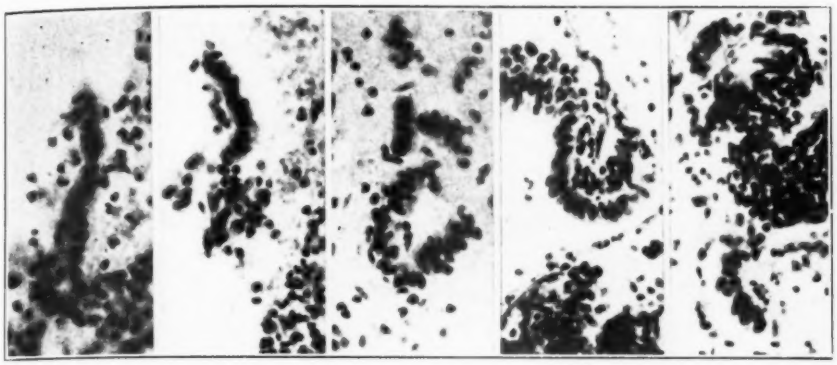
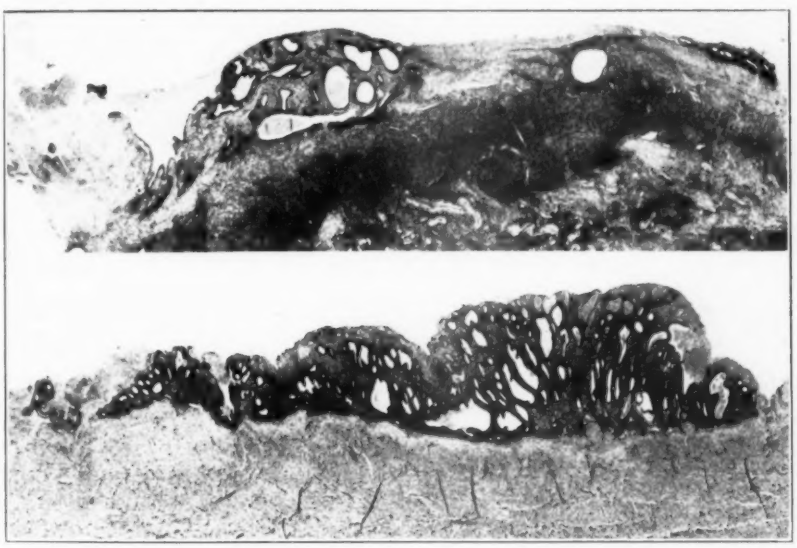


Fig. 47.—Five photomicrographs (x 250), the first two are of menstrual blood obtained from the vagina of two different patients. The third is from blood obtained from the uterus in curettage. The uterine epithelium in the first two appears just as healthy and just as much alive as that in the third. The fourth is from the menstrual blood of the uterine cavity (Case 3). The uterine epithelium stains as sharply as that in the third. The fifth is of a portion of the endometrial "cast" found in the tube of Case 3 and shown in Fig. 46. Epithelium, in the upper and lower ends of this photomicrograph, would seem to be alive. I have purposely photographed the healthiest appearing endometrial tissue that I could find in menstrual blood, the very kind I would choose to sow were I cultivating (implanting) this tissue.



Figs. 48 and 49.—Two photomicrographs (x 10), one of the endometriosis on the surface of the left ovary and the other of the endometriosis on the surface of the uterus adjacent to the ovary. Both are of the same structure and of the same apparent age. They must have had either a common origin or one must have been secondary to the other. They are in a situation (Fig. 43) readily "soiled" by material escaping from the abdominal ostium, of the patent left tube.

tubes. It would seem that during the menstrual life of women some substance escapes from the tubes into the pelvis which plays an important rôle in the etiology of pelvic peritoneal endometriosis, including the development of endometrial tissue on the ovaries. This substance may be menstrual blood in some instances and tubal secretions in others.

In either case epithelium may be present. Endometrial tissue on the ovary nearly always occurs on the *lateral* and *under surfaces* of that organ. These surfaces are usually in contact with the side of the pelvis, the posterior layer of the broad ligament, or the posterior surface of the uterus, thus forming a crevice between the ovary and these structures which would favor the retention of any material escaping from the tubes into the pelvis. In freeing the ovary containing an endometrial hematoma, the wall of the hematoma is usually torn or reopened at the site of a previous perforation of the cyst or where the endometrial tissue in the ovary first developed. The tear in the cyst caused by the operation is nearly always on either the *lateral* or *under surface* of that structure.

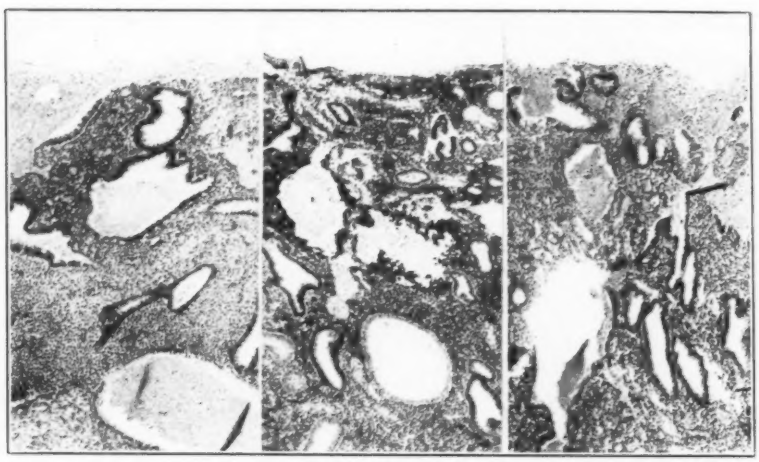


Fig. 50.—Three photomicrographs (x 40), the first of the endometrial tissue on the surface of the left ovary, the second of the uterine mucosa, the third of endometrial tissue on the surface of the uterus (Fig. 43). The histologic structure of all three is the same and they all show the same reaction to menstruation. As blood carrying with it bits of endometrial tissue escapes from the mucosa of the uterine cavity during menstruation, so might it likewise escape from these ectopic endometrial foci. Theoretically, it is possible that the blood in the tubes may have come from this source. It was, however, present in both tubes, was apparently coming from the uterus, and contained bits of endometrial tissue identical with those found in the blood of the uterine cavity. In addition, a moulded mass of endometrial tissue was found in this blood which conformed to a cast of the interstitial portion of the tube, through which it had apparently passed (Fig. 46).

Peritoneal endometriosis occurs most frequently in situations in the pelvis easily soiled by material escaping from the tubes and ovaries. It would seem that the tubes and ovaries are the chief distributing agents for the cause of pelvic peritoneal endometriosis. It is not peculiar to the pelvic peritoneum, as the appendix, cecum, small intestine and their mesenteries may be involved. The posture of mankind, whether standing, sitting, or lying down appears to be an important factor in determining the distribution of these lesions.

A comparative study of the lesions of peritoneal endometriosis with those of peritoneal carcinosis of implantation origin demonstrates that

the peritoneal reaction is the same in both instances. Endometrial tissue and cancer may grow on the surface of the peritoneum. Both may become embedded in the peritoneum by the tissues of the latter growing over them. Both may be included in mesothelial lined pockets and may be caught in adhesions.

THE ETIOLOGY OF ENDOMETRIUM-LIKE TISSUE IN THE OVARY

The endometrial tissue in a direct or primary endometriosis of both uterine and tubal origin shows a variety of lesions, such as typical endometrium with glands and stroma identical with that of the müllerian mucosa from which it came, and also dilated glands or cyst-like cavities lined by epithelium with very little or no characteristic endometrial stroma about it.

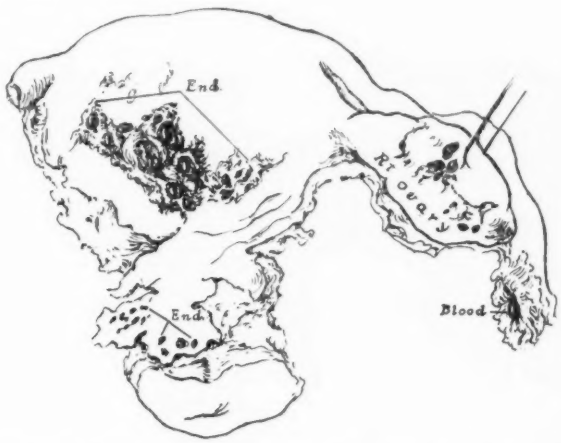


Fig. 51.—Posterior surface of the uterus, right tube and ovary (x ⅔). Case 1 of article. Uterus was retroflexed, and operation was performed on the second or third day of menstruation. Blood was observed escaping from both tubes on exposing the pelvic organs. Unfortunately, the distal end of the tube was not ligated and most of the blood in the tube was lost during the manipulation of the operation. Endometrial tissue was found in the tube (Fig. 53). Could the blood and the endometrial tissue in the tube have come from the ectopic endometrial tissue of the ovary and the posterior surface of the uterus? The endometrium-like tissue of the *lateral surface* of the ovary consisted of dilated glands filled with blood without any evidence of rupture. The endometriosis of the posterior uterine wall (Fig. 52) consisted of granulation tissue on its surface with deposits of endometrial tissue enmeshed in it and similar foci of endometrial tissue in the superficial portion of the uterine wall. Exposed endometrial tissue on the surface of the pelvic organs was not found. The endometriosis of the lateral surface of the ovary and the uterus are in situations readily soiled by material escaping from the tubes.

Marked changes in the mucosa lining the uterine cavity often occur. Of particular interest are those in the mucosa over a submucous leiomyoma. This mucosa becomes thin, the glands disappear, the stroma becomes less and less and in extreme cases the submucous leiomyoma is covered by a mucosa not unlike the mesothelial covering of a subserous leiomyoma. The endometrial epithelium in unfavorable conditions may be very similar to the peritoneal mesothelium and the surface epithelium of the ovary. Both the peritoneal mesothelium and the surface epithelium of the ovary, under the stimulation of any irritant, may become

hypertrophied and resemble the epithelium of the uterine mucosa. We recognize the mucosal covering of a submucous leiomyoma to be of endometrial origin, even though it simulates the peritoneal mesothelium and the epithelial lining of a follicular cyst of the ovary. When we find patches of typical endometrial tissue in an ovarian cyst with a lining similar to the mucosal covering of a submucous leiomyoma, can it be truthfully said that the typical endometrial tissue represents a metaplasia of the epithelium of a follicular cyst of the ovary, just because the cyst is situated in the ovary and portions of its lining resemble that of a follicular cyst? Is it not more logical to claim that the entire cyst is lined by one kind of tissue and that endometrial, portions of which have failed to attain their full growth, and thus present a histologic picture identical with the linings of some of the atypical endometrial

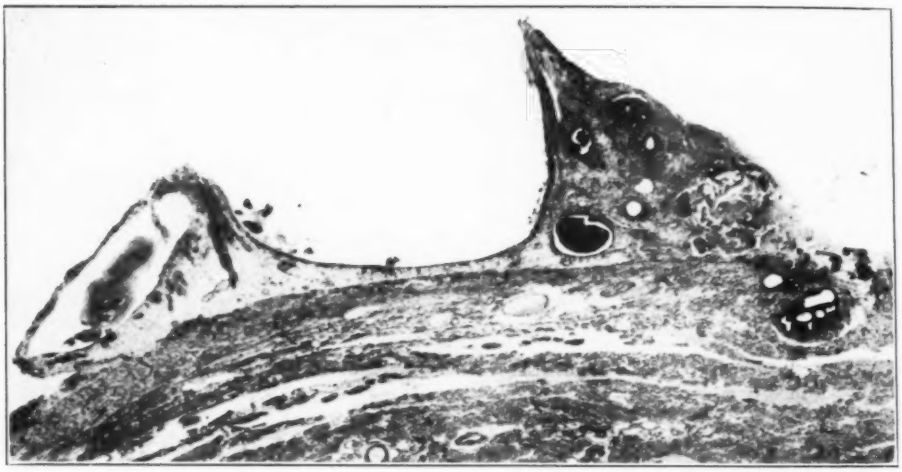


Fig. 52.—Photomicrograph (x 10) of a portion of the endometriosis of the posterior uterine wall. Granulation tissue, evidence of a reaction of the peritoneum to some irritant, has developed on the posterior surface of the uterus and has included in it endometrial tissue, just as cancer of implantation origin is included in the granulation tissue of peritoneal carcinosis. Endometrial "pockets" are also present in the superficial portion of the uterine wall. If this lesion is of implantation origin, it hardly could have come from the ovary. It is apparently an older lesion and could well be explained by the reaction of the peritoneum to material escaping from the tube; this material might contain uterine or tubal epithelium.

cavities of a direct endometriosis and the mucosal covering of submucous leiomyomas? See Figs. 59 and 60.

On account of the faculty of known endometrial epithelium to simulate peritoneal mesothelium and the surface epithelium of the ovary, it is often difficult to determine the origin of all misplaced endometrium-like tissue in the ovary.

Because a variety of epithelial structures develop in the ovary, it is natural to believe that müllerian tissue might also develop in that organ. Even if endometrial tissue arises on and in the ovary from the implantation of endometrial and tubal tissue on that organ, as I

believe it does, this does not exclude the origin of similar tissue from other sources.

THE ETIOLOGY OF PERITONEAL ENDOMETRIOSIS

I have stated in this article that I have encountered 293 patients with these lesions in the last five years. The question arises: are all these instances of true peritoneal endometriosis? I cannot prove this. In

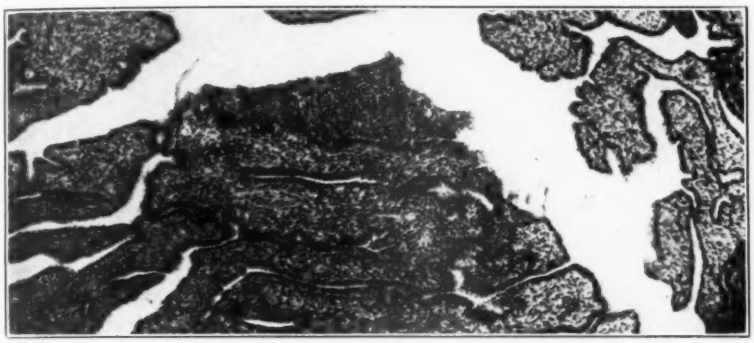


Fig. 53.—Photomicrograph (x 60) of a portion of the cross-section of the tube of Case 1. Endometrial tissue, consisting mostly of stroma infiltrated with blood and some epithelium, is adherent to the surface of the tubal mucosa. (The section unfortunately is thick.) This endometrial tissue resembled similar bits in the blood of the uterine cavity, from which the blood in the tubes was apparently coming.

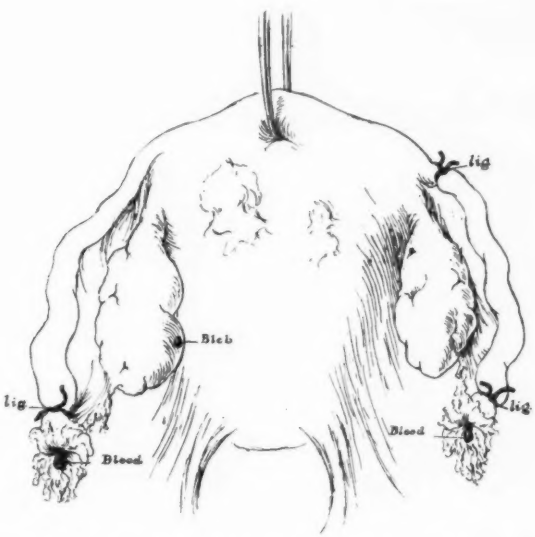
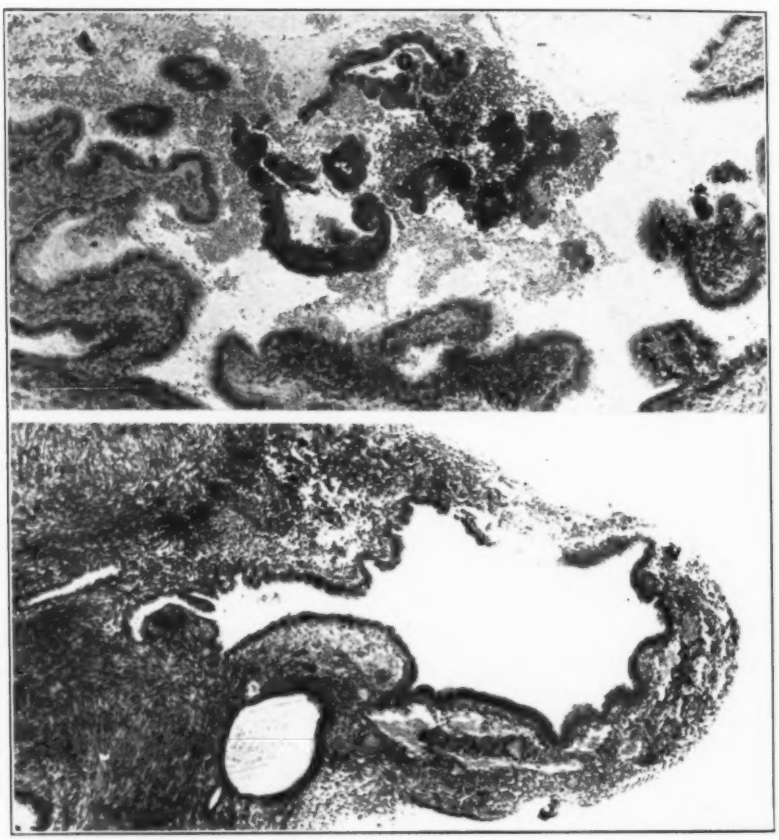


Fig. 54.—Uterus, tubes, and ovaries (x ½) of Case 2. The patient was menstruating at the time of the operation and blood could be seen coming from the abdominal ostia of both tubes. The distal end of the left tube and both the distal and proximal ends of the right tube were ligated before removing the uterus. The uterus, left tube and ovary, and the right tube were removed and hardened in formalin. Sections from both tubes showed blood and epithelial cells, a greater amount in the left tube as would be expected, because its proximal end, unfortunately, had not been ligated. Two small hemorrhagic blebs were found on the *under surface* of the left ovary, only one shown in the illustration. For the contents of the left tube and the structure of the larger bleb on the surface of the ovary, see Figs. 55 and 56.

many, typical endometrial tissue was found with a histologic structure identical with that of the mucosa of the uterus. In others, only atypical endometrial tissue was present, but similar to that often encountered in an endometriosis arising from the invasion of the uterine wall by its mucosa and by that of the tube. In still others, both typical and atypical endometrial tissue was found and one could trace the transition of one type of lesion into the other, just as one can follow a similar transition in the endometrial lesions of a direct endometriosis.



Figs. 55 and 56.—Two photomicrographs (x 60), the first of a portion of a cross-section of the left tube containing blood, strips of epithelium, a "gland" and fragments of *endometrial stroma* identical in structure and staining reaction with those present in the mucosa of the uterus, from which a larger amount of tissue had been cast off by menstruation. Compare with the ovarian lesion shown below. The endometrial tissue might have been forced into the tube during the operation, as the uterine end of the tube had not been ligated. Blood, however, was present in both tubes prior to the handling of the uterus.

The second photomicrograph (x 60) is of a section of the larger hemorrhagic bleb on the surface of the left ovary (Fig. 54). It is lined by epithelium of endometrial type with very *little stroma* and no *glands*. I was unable to find the loss of sufficient tissue to account for that in the tube, nor did the endometrium-like tissue of the ovary resemble that in the tube as closely as the mucosa lining the uterine cavity resembled it. It does not seem possible that the blood in the tubes (both) came from the small blebs on the left ovary (see Fig. 54).

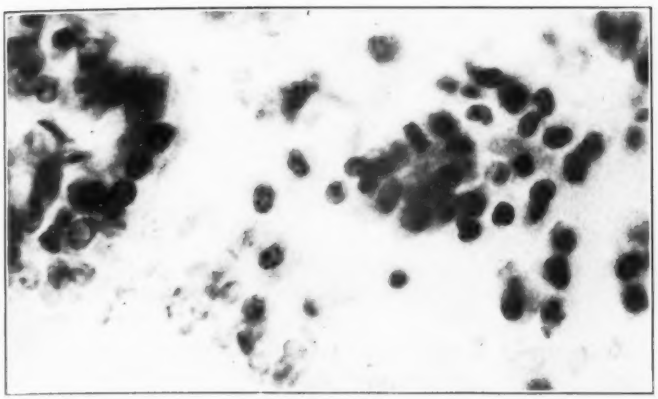


Fig. 57.—Photomicrograph (x 600) of a clump of epithelium-like cells in the lumen of the tube of a menstruating uterus (Case 4). Blood was observed escaping from the abdominal ostia of both tubes and apparently coming from the uterus. Peritoneal endometriosis was not present nor was there evidence of endometrial tissue in the ovaries. A clump of epithelium-like cells (to the right) is shown, surrounded by blood, in the lumen of the tubes. A mitotic figure is present in one of these cells. I am unable to state their origin. They certainly are not constituents of normal blood and they very closely resemble epithelium. Compare with the epithelium of the tubal mucosa to the left. Bits of uterine mucosa and clumps of epithelial cells were found in the blood obtained from the uterine cavity, but mitotic figures were not seen. If these are epithelial cells from the uterine or tubal mucosa they were alive at the time of the operation.

In four other cases blood was observed in the tubes of menstruating uteri at operation on patients without ectopic endometrial tissue in the pelvis, but the tubes were not removed. Films on slides were made of the blood obtained from the tubes by stripping them. Epithelium was found in these films and also cells which I was unable to identify. The trauma of stripping the tubes might dislodge tubal tissue and I have, therefore, discarded these. The blood in these cases apparently was coming from the uterine cavity and we know that, at times, it can carry with it bits of the uterine mucosa through the interstitial portion of the tubes, as shown by the bloody contents of tubes after curettage of the uterus.

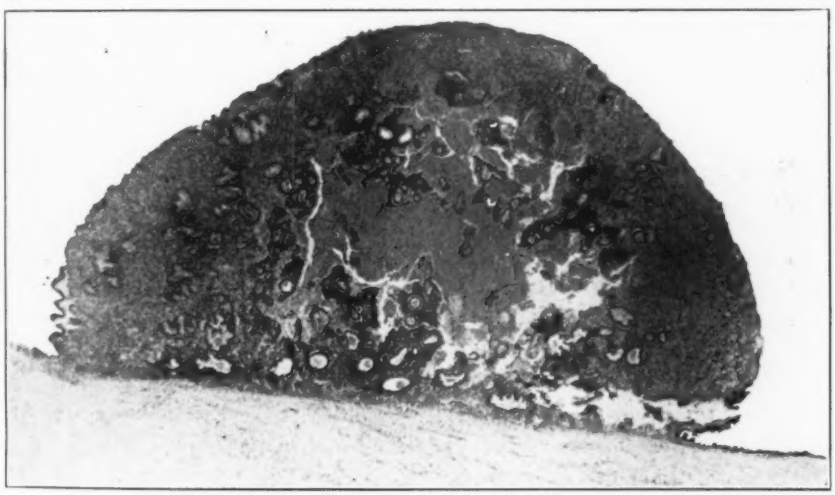


Fig. 58.—Photomicrograph (x 20) of a patch of "menstruating" uterine mucosa in the lining of the tube. Uterus, left tube and ovary were removed for uterine bleeding due to multiple leiomyomas; the patient was flowing at the time of the operation. The right tube and ovary were normal. A hematosalpinx was present on the left side adherent by its closed distal end to the ovary. Implantation-like lesions of endometrial tissue were present on the surface of the ovary adherent to the tube and in the wall of the tube. The blood in the tube evidently came from the endometrial tissue in the lining of the tube. The peritoneal endometriosis may have arisen from the same source, if the endometrial tissue was present in the tube prior to the closure of its fimbriated end. What is the origin of this patch of endometrial tissue? Is it misplaced endometrial tissue of congential origin, or a metaplasia of the tubal mucosa due to the influence of the ovarian hormone? Or even an implantation and subsequent growth of bit of uterine mucosa carried from the uterine cavity into the tube during menstruation or curettage? The patient had been curetted three years before.

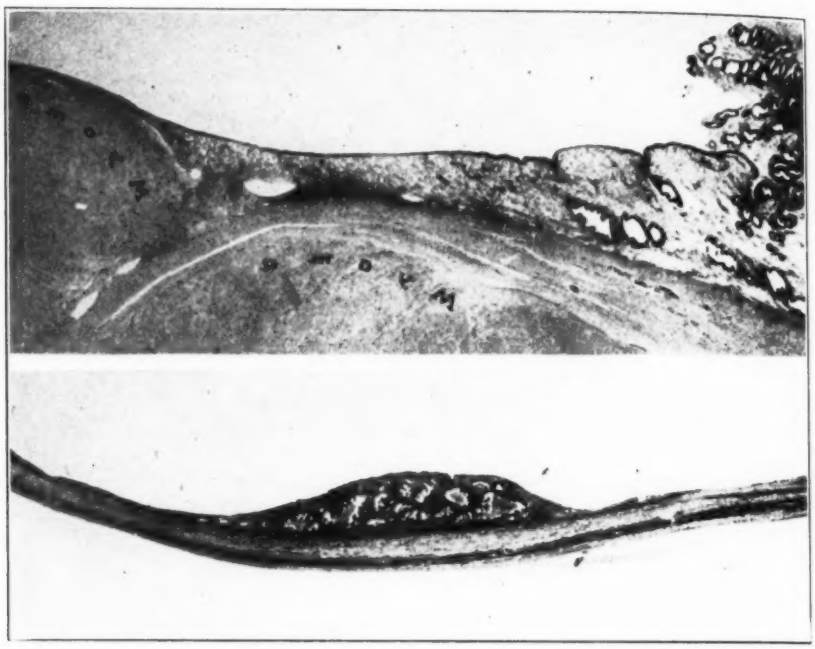


Fig. 59.—Two photomicrographs (x 10): the first of the uterine mucosa over a submucous leiomyoma and the second of the wall of an endometrial cyst of the ovary. The mucosa over the myoma is thin, there is very little stroma, the glands have disappeared and the surface epithelium is low. The patch of endometrial tissue in the lining of the ovarian cyst has the same histologic structure as that of the mucosa of the uterus removed with the cyst. The lining of the cyst on either side of the endometrial tissue has the same structure as that of the uterine mucosa over the myoma (Fig. 60). Is it a follicular cyst of the ovary with patches of endometrial tissue due to a metaplasia of its lining or an endometrial cyst, in which the greater portion of its lining had failed to reach its full growth? I believe the latter.

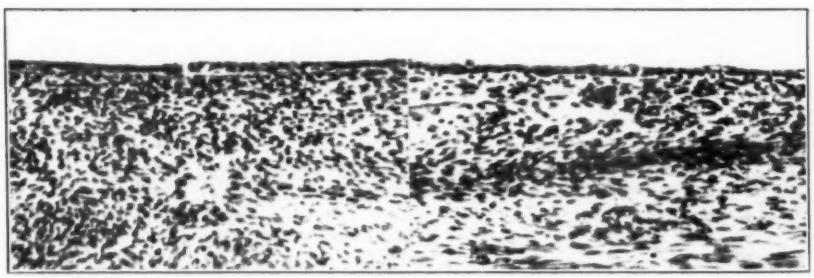


Fig. 60.—Two photomicrographs (x 130), the first of the uterine mucosa over the submucous myoma and the second of the lining of the ovarian cyst adjacent to the patch of typical endometrial tissue. Their histologic structure is the same.

The peritoneal mesothelium, when stimulated by any irritant, may assume an epithelium-like structure. Gland-like inclusions of the mesothelium may arise and also cavities lined by it. Should the irritant contain blood or cause bleeding, the conditions resulting from this might be incorrectly attributed to a menstrual reaction.

Even if peritoneal endometriosis arises from the implantation of endometrial and tubal tissue on the surface of the peritoneum, as I believe

it does, this does not prove that all instances of endometrium-like tissue involving the peritoneum arise from this source.

SUMMARY

Menstrual blood escapes into the peritoneal cavity from (1) the rupture or perforation of endometrial cysts or cavities of the ovary and possibly of other pelvic structures; (2) menstruating endometrial tissue growing on the surface of the ovary and other pelvic structures; (3) the uterine cavity as a back flow through the tubes; (4) menstruating tubal mucosa.

Menstrual blood, irrespective of its source, at times, contains bits of endometrial tissue set free by menstruation.

Endometrial tissue disseminated by menstruation is sometimes alive and will continue to grow, if transferred to situations suited to its growth.

The peritoneum and surface of the ovary are suited to the growth of endometrial tissue.

The lesions of peritoneal endometriosis often occur in situations and under conditions indicating (at least suggesting) their origin from menstrual blood escaping from the above mentioned sources.

The local reaction of the peritoneum to the endometrial tissue in peritoneal endometriosis is similar to the local reaction of the peritoneum to cancer in peritoneal carcinosis of implantation origin.

CONCLUSIONS

These studies indicate that peritoneal endometriosis sometimes arises from the implantation of endometrial tissue disseminated by menstrual blood escaping into the peritoneal cavity.

Endometrial and tubal tissue disseminated by other means may do the same. This phase of the subject will be considered in a later paper.

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180 Washington Avenue.

OVARIAN METASTASIS WITH CANCER OF THE UTERINE BODY. IS TRANSTUBAL IMPLANTATION AN IMPORTANT FACTOR?

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THERE is no question that a reciprocal relationship exists between the uterus and the ovary as regards the extension of cancer from one organ to the other. In many cases such considerations as the size and exact location of the tumors, the clinical history, and the histologic examination make the determination of the primary source of the disease comparatively simple. In other cases it may be difficult or perhaps impossible to settle this point satisfactorily. The ovary is an organ which is notoriously prone to the secondary deposit of cancer tissue, not only from other pelvic organs, but even more from such distant locations as the stomach, intestinal canal, gall bladder, breast, or thyroid. It is a striking exception to the old dictum of Virchow, that an organ which is prone to primary cancer is rarely the seat of a metastatic cancer, and vice versa.

When uterine cancer and ovarian cancer coexist, either organ may be the primary seat of the disease, or, in rare cases, it is even possible that the two cancers may arise independently of one another. A considerable group of cases in the literature have been interpreted in this last named way, but, in the very nature of things, they must be comparatively rare. The conclusion as to the independent origin of two such tumors has usually been based chiefly upon the difference in type of the two tumors. When dealing with the ovary, however, this would seem to be a hazardous criterion, for there is no organ in the body in which the local environment appears to exert such a profound modifying influence upon the invading cancer as the ovary. This is well illustrated in the well-known Krukenberg tumor, which is secondary usually to a pyloric or other gastrointestinal cancer, and which, nevertheless, presents a microscopic picture not in the least suggesting the usual pyloric cancer.

As to whether the ovary or the uterus is more frequently the primary source in cases of coexistent disease of these organs, opinion is somewhat divided. Pfannenstiel, Werner and others believe the ovary is more often the primary seat. Werner, for example, in his series of 15 cases, considered the ovary to be the primary seat in 6, and the uterus in 4. Three he believed to arise independently, and the other 2 he classed as doubtful. Others feel that the uterus is

more often the primary seat, and this is more in accord with our ideas as to the channels of spread of the disease.

This paper will be limited chiefly to a consideration of the means of extension of uterine cancer to the ovary. The material upon which the study is based is a series of 147 cases of adenocarcinoma of the uterus studied in the laboratory of Gynecological Pathology at the Johns Hopkins Medical School. Of this number 7 were found to show extension to one or both ovaries. This group does not, of course, include cases of combined cancer which were interpreted as primarily ovarian, in which, for example, an advanced carcinoma of the ovary was associated with cancer deposit in some part of the uterine wall.

There are four routes to be considered in discussing the propagation of cancer from the uterus to the ovary, viz.: (1) the lymph stream; (2) the blood stream; (3) direct extension, and (4) implantation of cancer material transported through the tube.

The Lymphatic Route.—The accepted channel of dissemination of cancer in any part of the body is the lymph stream. This, of course, is not a mere assumption, for this method of spread has been incontrovertibly demonstrated by microscopic study. Whether one believes in the importance of the permeation theory of Handley, or whether one accepts the possibility of transportation to a distance of detached cancer cells, as most authorities do, is beside the point. Certainly no one today will deny that cancer of the breast, or of the stomach, or of the lip, or of the cervix is spread by the lymph route above all others. It is, indeed, almost the only one considered by the surgeon in planning his operative attack upon the disease. This same view has been held by most gynecologists and pathologists with regard to the dissemination of cancer of the uterine body.

The lymphatic supply of the corpus uteri has been exhaustively studied by a number of investigators, particularly Poirier, Sappey, Bruhns, and Kroemer. In a general way the lymphatics arising from the uterine body are arranged in four chief groups, as follows: (1) those proceeding from the lower and middle parts of the corpus to the hypogastric glands at the iliac bifurcation, chiefly to the upper group of these glands; (2) those proceeding with the ovarian vessels between the tube and ovary, receiving in their course the ovarian lymphatics, and terminating in the lumbar glands in front of the lower aorta; (3) those extending between the fundus and the walls of the fallopian tube, and (4) those extending from the fundus, along the round ligaments, to the inguinal group of glands.

This distribution of the lymphatics, to my mind, explains adequately almost all the metastases which occur with corporeal cancer, including those which may be observed in the ovaries. For that matter, I believe that it likewise explains the spread of cancer from the ovary to the uterus, although in this case, recourse must be had

to the doctrine of retrograde lymphatic dissemination. This doctrine, however, is accepted by all pathologists, and its possibility is easy to understand. The lymph pressure is extremely low, much lower than that even in the small veins. The lymph circulation, therefore, is a very leisurely one. Cancer cells entering a lymph radical are not swept away quickly, as when they enter the blood stream, but they saunter along slowly, and are easily deflected into bypaths by the slightest obstruction ahead.

Cancer cells from the ovary may thus be deflected back towards the uterus. Careful examination in such cases will often show emboli of cancer cells in the lymphatics between the ovary and tube, as I have myself observed. Similar findings are reported by Kroemer and others.

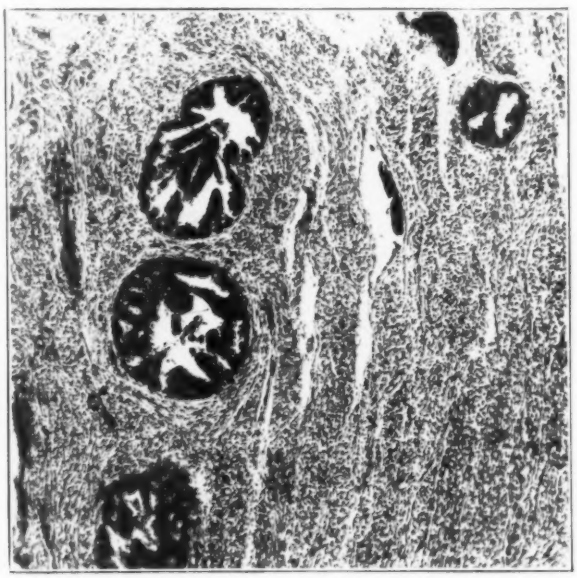

Fig. 1.—Cancer metastasis in myometrium with the primary growth in the ovary. The endometrium in this case showed no trace of cancer, nor did the peritoneal surface of the uterus. The metastasis, I believe, was due to lymphatic dissemination.

Cancer cells thus carried back to the uterus would first find lodgment in the perimetrium and myometrium, so that metastatic nodules would be looked for here rather than in the endometrium. This, as a matter of fact, is exactly what does occur in most cases I have observed. (Fig. 1.) Other authors have made the same observation. Werner, for example, believes that uterine metastases from the ovary occur, in the order of frequency, in the peritoneum first, then the myometrium, and then the endometrium. If this is true of the route from ovary to uterus, how much easier it is to accept the ready possibility of cancer cells from the uterus being carried along with the lymph

stream to the ovary, for this involves little or no deviation from the natural course of this lymph drainage.

Another fact which speaks strongly for the lymphatic route is the frequency with which the tube is involved, often without any trace of cancer in the ovary. In some of the reports, indeed, tubal metastasis, not apparently in the nature of simple extension, is found more often in the tube than in the ovary. In 3 of my 7 cases of ovarian metastasis, the tube was likewise involved. This is explained, no doubt, by the fact that there is a very direct route for the cancer cells from the fundus to the tubal wall. It is of interest to note, also, that

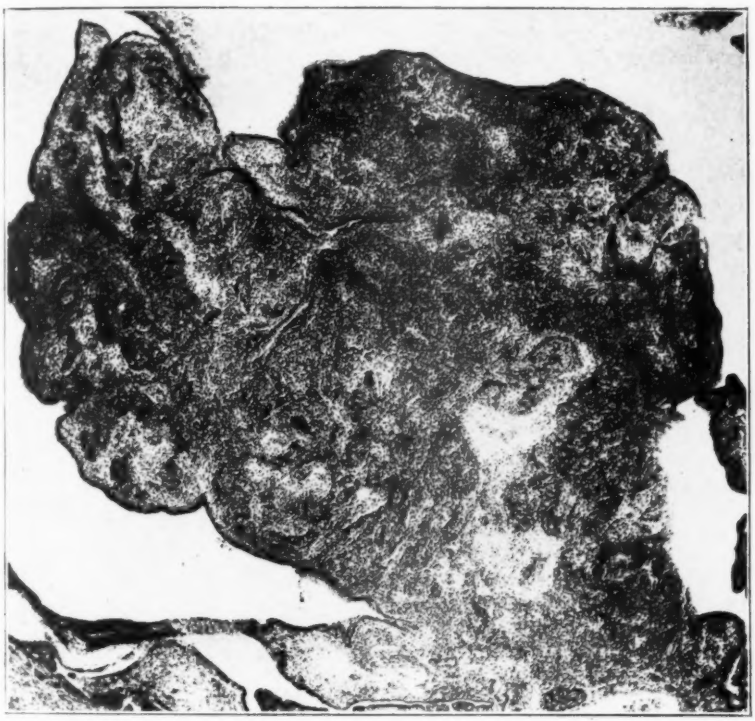

Fig. 2.—Cancer metastasis in tube, in a case of adenocarcinoma of the fundus. There was ovarian metastasis as well. The epithelium of the tube appears intact, and the assumption is that the metastasis was due to lymphatic deposit of cells from the uterus. Note the plugs of cancer cells in the lymphatics in the lower portion of the section.

the tubal metastases most often occur in the tubal wall rather than in its mucosa, as one might expect if the tubal involvement were due to actual implantation of tissue transported out from the uterus. (Fig. 2.) Often, indeed, the mucosa is perfectly normal, although, as the tubal cancer grows, it pushes into the tubal lumen, so that its original site cannot be determined. There is, of course, no reason why a tubal metastasis from the uterus, or a uterine metastasis from the ovary, could not occur in the mucous membrane of either of these

organs, for both the endosalpinx and the endometrium are richly supplied with lymphatics. The lesser frequency of metastasis in the mucous membrane, as compared with that in the muscle, is apparently due, as already stated, to the fact that the cancer cells usually reach the mucosa only after running the gauntlet of the muscularis.

Of the 7 cases in my series in which adenocarcinoma of the uterus had extended to the ovary, all except one, it seems to me, are logically explained on the basis of lymphatic dissemination. Even this one exception, as I shall discuss later, may be apparent rather than real. In Case 1 there was a typical corporeal cancer, with involvement of one ovary, which, however, was perfectly smooth and only

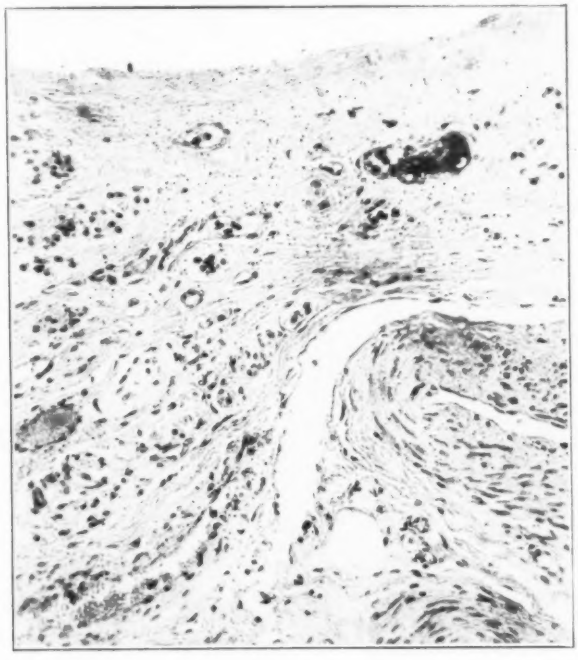

Fig. 3.—Cancer cells in lymphatics of the mesosalpinx, in a case of adenocarcinoma of the uterus with metastasis to the ovary.

slightly enlarged. Its surface showed no implantation, but cancer was found in the deeper portions of the ovary. Furthermore, plugs of cancer cells could be demonstrated in the lymphatics of the mesosalpinx. (Fig. 3.) The tube showed carcinoma in its wall, although the mucosa was normal. This patient was fifty-five years old.

Cases 2 (age sixty) and 3 (age forty-six) are almost identical in the distribution of the disease, except that in both of them the ovarian cancers were larger. In both of them, however, the ovarian surface was smooth, and in both of them cancer emboli could be demonstrated in the lymphatics of the broad ligament. In Case 4 (age fifty)

the uterine cancer was associated with a small cancer nest in one ovary, which grossly appeared normal. This cancerous area was not on the surface, as one might expect if the involvement were the result of implantation, but near the hilum, as one would expect if the cancer had been imported by way of the lymphatics (Fig. 4).

Case 5 (age fifty) was characterized by involvement of both the left tube and left ovary, the ovarian tumor measuring 6 by 4.5 by 2 cm. Incidentally, both tubes were completely sealed by advanced inflammatory disease, the left tube forming a hydrosalpinx 3 cm. in diameter. This fact would seem to have precluded the possibility of transportation of cancer material though the tube from the uterine

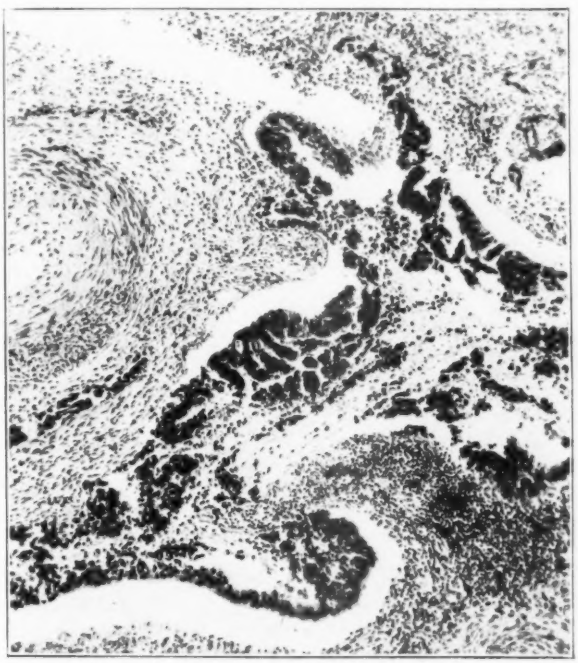

Fig. 4.—Small cancer metastasis, apparently of lymphatic origin, in the hilum of the ovary. The organ was not enlarged, and its surface was perfectly smooth. Only this one small cancer nest could be found in the ovary. There was an adenocarcinoma of the uterine body.

cavity. Cancer plugs were found in the lymphatics of the tubal wall, although this would be expected because of the presence of a definite cancer focus in the tube. In Case 6 (age forty-seven), finally, there was an adenocarcinomatous growth of rather small size (3.5 by 2 by 1 cm.) in the uterus, near the right cornu. The left ovary measured 3.5 by 2.5 by 2 cm., and its external surface was smooth and uninvolved. On section, a friable area was seen near the hilum, and this was shown microscopically to be cancer. The right ovary was the seat of a multilocular cystadenoma about 12 by 6 by 6 cm. in size,

with a twisted pedicle. Near the hilum of this ovary was a friable cancer area 2.5 by 3 cm. in diameter, with a structure similar to that seen in the other ovary. To explain such a case on any other basis than that of lymphatic dissemination would seem far-fetched.

I have laid some stress on the fact that in all these cases the surface of the ovary was smooth and uninvolved, as I do not see how this condition could obtain if cancer had planted itself on the surface.

Blood Vessel Route.—While cancer is believed to be disseminated chiefly by the lymphatics, there is no doubt that the disease may at times be propagated by the venous blood stream. This has been abundantly shown for certain types of cancer, as, for example, the not infrequently distant transportation of hypernephromas arising in the kidney. Arzt has demonstrated a similar route in a case of ovarian cancer which secondarily involved the uterus. As regards ovarian metastases of uterine cancer, Ribbert believes that the blood stream is a more important channel of distribution than is the lymph circulation, although this view is not generally accepted. Certainly there is very little direct evidence bearing on this point. As in other tissues, however, there is no doubt that at times the cancerous process penetrates into venous channels, so that the cancer cells are readily disseminated, perhaps to distant points. For the occasional distant metastases, there would seem to be no other plausible explanation.

Direct Extension.—The progressive growth of a cancer in the uterus is associated with an increasing penetration into the surrounding tissues, this being primarily due to a multiplication of the cells, much as this might occur in tissue cultures. Around the original cancer site there is thus produced a halo of cancer projections which destroy the normal tissue in their growth. Synchronously with this, of course, there proceeds a growth along the lymphatic channels, and a breaking off and deportation of cancer particles for either a short jump or a long journey. In advanced carcinoma of the fundus one will often see cancer masses invading the uterine wall throughout its thickness, so that peritoneal nodules are often produced. In cases in which the ovaries are adherent to the uterus, they may become involved by direct continuity. It is, of course, difficult at times to draw a sharp distinction between direct extension and lymphatic propagation, as already stated. For example, Milner has demonstrated definite lymphatics, containing cancer cells, in the adhesions between a cancerous uterus and the ovary which had become involved, apparently by direct penetration of the cancer through the uterine wall.

In one of my own cases (Case 7, age 52), in which the surface of an adherent ovary is involved in the cancer, my interpretation of the process would be the same. In this case, the uterine cancer was extensive, involving not only the entire thickness of the uterine wall,

but the cervix and the broad ligaments as well. This, to my mind, is a more logical explanation than to attribute the ovarian cancer to the growth of cancer tissue transported from the uterus through the tubal lumen. This case is the only one of my series which shows any surface involvement of the ovary, although, I may add, there was marked invasion of the deeper portions as well.

Implantation.—In going over our cases I have been especially interested in attempting to find out how many could be considered to have arisen, with any great degree of probability, from the implantation and growth in the ovary of cancer tissue carried out from the uterus by way of the tube. The incentive for the study was, of course, the paper of Sampson, in 1924, in which this author expressed the view that such a mechanism was probably not uncommon. He reported 4 cases which he interpreted in this way. Furthermore, on the basis of this work, he made two important practical suggestions. The first of these was the advice to ligate the fimbriated extremities of the tubes before proceeding with the removal of the cancerous tissue. The second was a warning to abstain from preliminary curettage unless absolutely necessary, for fear that portions of the curetted tissue might make their way out through the tubes into the peritoneal cavity.

In a paper presented before this Society at its last meeting, I submitted certain evidence tending to cast doubt upon Sampson's views as to the importance of retrograde transportation of endometrial tissue at the time of menstruation, as a cause of pelvic endometriosis. In that paper I alluded also to the incompleteness of his evidence for a similar mechanism in the production of ovarian metastases from cancer of the uterine body. The present study has served to fortify my views on this point.

In the first place, ovarian metastases are a relatively infrequent accompaniment of uterine cancer. In my own series of 147 cases of carcinoma of the body, many of them far advanced, only 7 cases of ovarian cancer were found. Norris and Vogt, in their 115 cases of corporeal cancer, found ovarian involvement in only 2, while the tube was involved in 1. In the 44 cases of Meigs there were 5 with carcinoma in either the tubes of ovaries. Schottländer and Kermauner found only 3 instances of ovarian metastasis in 140 cases of adenocarcinoma of the uterus.

Sampson explains the infrequency of ovarian metastasis as due chiefly to the fact that the great majority of cases of adenocarcinoma of the uterus occur in women well beyond the menopause, which is undoubtedly true. But a not inconsiderable proportion are still actively menstruating. For example, fully 33 of my 147 cases occurred in women not over fifty years old, and fully 13 in women not over forty-five. Thirty-three of Norris' and Vogt's 115 cases were below

fifty. In my 7 cases of ovarian extension of uterine cancer only 2 were in the menstruating age. This would seem to speak against the theory of Sampson that regurgitated menstrual blood may easily carry with it cancer particles that reach the ovarian or peritoneal surface and implant themselves.

Furthermore, even aside from the factor of menstrual bleeding, every advanced case of uterine carcinoma is associated with bleeding into the uterine cavity, which, if Sampson's theory were correct, could be readily regurgitated into the peritoneum.

This would theoretically be facilitated by the fact that colicky contractions of the uterus not infrequently occur when there is an intrauterine growth of considerable size, especially as the passage-way toward the vagina is often impeded. And yet, in spite of all these considerations, cancer extension to the ovary is exceptional. Furthermore, if tubal regurgitation were frequent, one would expect to find implantations, not only on the ovarian surface, but also on the peritoneum in the immediate vicinity of the fimbriated end of the tube. Such, however, is not the case, as I shall emphasize later in this paper.

In a more recent paper, Sampson has expressed the view that endometriosis is in some cases due to the dissemination of embolic particles of endometrium which enter the uterine sinuses at the time of menstruation. It is possible that this may at times occur. Indeed, I had suggested such a possibility in a discussion of the paper presented by Sampson before this Society, in 1925. I question, however, whether this factor is of importance in the explanation of the cause of peritoneal or ovarian endometriosis in general. If the uterine sinuses at the time of menstruation gave ready admission to endometrial particles, would they not just as readily give entrance to the particles of cancer when that disease is present in the endometrium? And would not such cancer cells be disseminated far and wide along the blood channels? But hematogenous dissemination is rare with corporeal cancer, which, like most cancers, travels by way of the lymph channels. It would seem that there must be some protective mechanism present in the uterine wall to guard against the invasion of the blood and lymph stream which might be expected to occur because of the casting off of the endometrial surface at each menstruation and the consequent opening up of blood vessels and lymphatics.

In his discussion of the subject in 1924, and his description of the 4 cases upon which his views were apparently based, Sampson did not even mention the possibility of a lymphatic origin for the ovarian cancers, although this is the one which would naturally occur to most of us in explaining the metastases in these cases. Certainly this route cannot be excluded, and, as this is the recognized route for cancer dissemination, it cannot be dismissed without much stronger evidence than is contained in Sampson's paper. I shall not analyze these cases,

but I believe that most pathologists, on the premises which his case reports give, would conclude that the cancer had been disseminated through the usual lymphatic channels. In some of my own cases, which have been quite similar to those of Sampson except for the admixture of sarcoma in one or two of the latter's cases, cancer cells can be actually seen in the lymphatics of the tube or broad ligament.

It would seem that Sampson has attached too much importance to the occasional findings of free cancer particles in the lumen of the tube. Such a finding is becoming increasingly frequent as we search for it more intensively, but there is as yet no justification for considering that these broken off particles are responsible for the extension of the growth. Some of these particles—in my opinion, most of them—are moving downward toward the uterus rather than upward toward the peritoneum.

Certainly where there is a vegetative growth present on either the uterine or the ovarian surface it would be strange if particles of the friable tissue did not, through sloughing or possibly through some form of trauma, break off and migrate. This may happen with cancer in any part of the body. It undoubtedly occurs with cancer of the pylorus, the broken off particles passing into the intestine. But metastasis occurs not along the line of tissue migration, but along the lymphatic channels. The same process occurs with fungous cancer of the cervix, in which case the particles are expelled through the vagina. But, here again, the metastases follow the distribution of the lymphatics. These examples might be multiplied.

In the case of corporeal adenocarcinoma, the path of the broken off tissue, in the event of its being forced backward along the tube, happens to parallel the route of the lymphatics, and hence the distinction cannot be made so sharply. But, as I have already said, there is no justification for assuming a special mechanism here until more definite evidence is produced. I shall not again review the question of whether or not retrograde transportation of tissue through the tube actually occurs with any great frequency, as this was discussed in my former paper. I may merely add that such a backward propulsion would seem especially hard to conceive of in the usual body cancer case because we have to deal with a senile, contracted tube which has probably lost any peristaltic activity that it might have possessed in earlier life. Furthermore, the factor of menstrual regurgitation, to which Sampson attaches so much importance, is no longer operative at this age.

The whole subject of implantation cancer is apparently viewed with an increasing skepticism by pathologists. More and more evidence is being brought forward in favor of the view that many instances of what was formerly looked upon as implantation are really due to lymphatic dissemination. For example, carcinoma of one lip of the

vulva may at times be associated with a "contact infection" of the other lip. These are now looked upon as being due to lymphatic metastasis. Again, as MacCallum says, the carcinoma nodules which may be observed in the pleura with carcinoma of the lung, and which were formerly explained on the implantation theory, have been definitely shown to be continuous with underlying cancer-containing lymphatics. Sampson believes that cancer particles may implant themselves on the mucous surface of the tube. Such a possibility seems very remote, if one may judge from general pathologic literature.

One of the most interesting gynecologic examples of supposed implantation of cancer is seen in the not infrequent association of pyloric and ovarian cancer. In these cases the popular explanation has been that cancer cells from the pylorus have been sifted down, aided by intestinal peristalsis and perhaps by the movements of the omentum, until they come into contact with the ovary, where they plant themselves and grow. But in many of these cases the pyloric cancer is small, and there is no demonstrable penetration to the serosa.

Again, why do the cancer cells, if they actually do enter the peritoneum, gravitate all the way to the ovary, leaving undisturbed the peritoneum over the intestinal and other viscera, although even an intact peritoneum offers a very suitable nidus? The germinal epithelium is really thicker and probably more resistant than the peritoneum elsewhere, and certainly the tunica albuginea is a dense and not a very vascular tissue. Ovulation, of course, makes a break in the ovarian surface, but this break is quickly repaired, probably within a matter of hours.

Moreover, the ovarian cancer which is rather characteristically seen in association with stomach cancer has a smooth surface, and retains the general shape of the ovary. It shows no surface growth, as would be expected if the cancer were planted from the outside, and, indeed, often shows a definite outer capsule. These and other considerations are difficult to reconcile with the implantation theory.

A more plausible explanation, to my mind, is the one based on the assumption of lymphatic dissemination. The lumbar glands, which receive the upper uterine and ovarian lymphatics, are in close communication with the retrogastric glands, which are involved in most cases of pyloric cancer. Amann reports finding the lumbar glands enlarged, presumably from cancer, in several cases of ovarian cancer associated with pyloric cancer. So far as I have been able to learn, a complete demonstration of this route of dissemination has not been made, probably because of the inherent difficulties of the problem. It would mean a serial section study, an almost impossible task under the circumstances.

It would seem difficult, on the implantation basis, to explain the characteristically bilateral involvement of metastatic ovarian cancer of this type. On the other hand, since the lymphatics of both ovaries terminate in the lumbar group of glands, such a bilateral involvement would seem quite natural.

Much of what has just been said as to ovarian carcinoma which is secondary to pyloric cancer, applies with equal force to those cases which are associated with adenocarcinoma of the corpus uteri. If implantation of transported material were a common cause, one would expect to find ovarian metastases much more frequently, and especially in women during menstrual life. But they are rare at any age, and especially rare during the reproductive epoch, as I have already indicated. Furthermore, they are characteristically bilateral, a fact which suggests a metastatic extension from a central distributing focus, such as the lumbar glands, rather than a simple accidental implantation. Again, if cancer material from the tube were carried out into the peritoneum, one would expect to find, more frequently than is the case, carcinomatous growths on the peritoneum near the fimbriated extremity of the tube, perhaps even in relatively early cases of body cancer. As a matter of fact, peritoneal carcinosis, rather rare at best, almost never occurs until the disease is in an advanced stage, usually not until it has penetrated to, or almost to, the peritoneal coat of the uterus. It is not rare, even in such advanced cases with peritoneal metastases, to find the ovaries normal, although theoretically they would be the first stopping place of cancer material escaping from the tube.

For the reasons which I have indicated I believe that there is little evidence to indicate that implantation of transported cancer material plays a rôle of any great importance in the production of ovarian metastases. Indeed, so far as I have been able to find, it has not as yet been indisputably established in a single case. The mere finding of free cancer particles in the tubes, as I have already indicated, cannot be considered as evidence that a cancer of the ovary has been produced by such migration of tissue. This would be "post hoc, ergo propter hoc" reasoning of the worst sort. Certainly, from what we know of cancer dissemination generally, one is not justified in assuming an implantation origin of ovarian metastasis unless the well-authenticated lymphatic mechanism can be excluded.

I do not, of course, mean to apply that cancer implantation cannot occur under any circumstances. In the peritoneal cavity, at any rate, the general dissemination which is not infrequently found with ovarian carcinoma is commonly explained on the implantation basis, and it is quite possible that this view is correct. In a broad sense, indeed, the peritoneal cavity is a magnified lymph space, and peritoneal implantation of cancer is thus easier to conceive of than implantation

upon an intact mucous surface. The other example of implantation which suggests itself to the operating surgeon is the variety which may be observed, though rather rarely, in the scars of operative incisions. One is therefore not justified in denying the possibility of implantation altogether, although, as already stated, the occurrence in general is being viewed with increasing skepticism by pathologists.

From what has been said in this paper, it need scarcely be added that I do not feel convinced of the value of Sampson's recommendation to ligate the tubal ends in all cases in which a cancerous uterus is to be removed, or the wisdom of his advice to abstain from preliminary curettage except in very doubtful cases. As regards the first of these procedures, it is, of course, hard to find fault with any bit of technic which adds in even the slightest degree to the safety of any operation. Whether valuable or not, there is certainly no harm in this procedure, and it is possible that future investigation will show that it has a very definite value. From the evidence thus far available, however, I question whether this operative step will improve our operative results very materially, except of course where there is associated tubal cancer. Here it would seem to be a valuable means of preventing a squeezing out of cancer material from the tube. In the ordinary case, in which the tubes are perfectly normal, or at any rate, free from cancer, the value of tubal ligation will not be great.

It is far more important, to my mind, to guard as far as possible against squeezing cancer cells into the lymphatics by improper manipulation of the uterus. The early application of clamps to the broad ligament will do much to prevent such operative dissemination of cancer cells. Clamps applied vertically, at each side of the fundus, moreover, will furnish a convenient means of traction upon the fundus. If a direct means of elevating the uterus is desirable, the use of a heavy traction suture through the fundus is preferable to the compressing type of uterine traction clamp sometimes employed. The latter would certainly seem capable of squeezing cancer cells into the lymphatics of the uterine wall.

To repeat, therefore, while there can be no objection to tubal ligation, the evidence at hand would seem to indicate that this procedure is not nearly so important as the employment of means to avoid lymphatic dissemination of cancer cells at the time of operation.

Speaking of the dangers of diagnostic curettage as a preliminary to cancer operation, Sampson states that he uses diagnostic curettage only in those in whom he does not suspect cancer or when he considers the patient a poor operative risk. In all other cases of suspected cancer of the body of the uterus he removes the uterus without a preliminary curettage. This, to my mind, is an unwise and dangerous teaching. It impresses one as having even less justification than the advice to refrain from diagnostic curettage because of the

danger of lymphatic dissemination. Certainly there is at least some evidence to suggest that possibly curettage in cancer, like incision into a malignant breast tumor, may lessen a patient's chances for cure. And yet, as Frank has well emphasized, nearly all gynecologists practice this method. There are two reasons for this. One is that the evidence as to the dangers of this gynecologic procedure is as yet unconvincing, if one may judge from such reports as that of Norris and Vogt, in which the final results are compared in cases curetted and those not curetted. The second and probably more important reason is that it is only by microscopic examination of the scrapings that we can, in a large proportion of cases, make the diagnosis, and thus avoid grave abdominal operations in many cases in which mere symptomatology might seem to justify them. There are, however, certain cases in which the omission of curettage is justified; for example, persistent and perhaps profuse uterine bleeding in women well beyond the menopause. But in cases of bleeding of slighter amount or shorter duration even in old women, and especially in suspicious cases in women who have not yet passed the menopause, the diagnosis is usually absolutely impossible without the aid of the microscope; i.e., without diagnostic curettage.

In many cases where I had suspected adenocarcinoma I have been agreeably surprised to find no evidence of cancer under the microscope. Furthermore, from the examination of tissue obtained by other surgeons under similar circumstances, I know that this is a common experience. To have removed all these uteri would have meant an unnecessary mortality,—not inconsiderable even with expert operators, and much more serious in the hands of less experienced surgeons who might follow this advice. Even, therefore, if we were certain that diagnostic curettage were associated with a definite risk of dissemination, most of us would choose this in most cases in preference to the greater risk of performing panhysterectomy unnecessarily. Furthermore, it is to be remembered that the cases in which radical operation is justifiable without preliminary curettage, are obvious ones, of long duration. The early cases, however, for which we should be always on the alert because of their ready curability, cannot be diagnosed except by microscopic examination of the curettings.

All that has been said as to the justifiability of diagnostic curettage in spite of a theoretical danger of lymphatic dissemination applies with far greater force to the performance of preliminary curettage, in spite of the theoretic danger of implantation from cancer tissue escaping through the fimbriated extremities of the tubes into the peritoneal cavity. The evidence for the latter danger is as yet very incomplete. In fact, it is perhaps not too much to say that it has not been conclusively demonstrated in a single case. The occasional oc-

currence of menstrual regurgitation through the tubes—for I believe it is only occasional—and the finding of bits of endometrial or even cancer tissue in the tubes is not conclusive, as I have already emphasized.

The ideal method of management of cases of suspected adenocarcinoma will, I believe, obviate any danger of either lymphatic or tubal dissemination, and, in addition, it possesses other valuable advantages. Diagnostic curettage should be done, but it should be followed at once by the radical operation if cancer be found, and not after an interval of from one to many days. This method depends, of course, upon a proper frozen section technic, which should be a part of the equipment of every operating room. It is only rarely that an accurate diagnosis cannot be made from the frozen section, for with adenocarcinoma, even more than with other types, the diagnosis is commonly made from the microscopic pattern rather than from the study of cell changes.

Once the diagnosis is made, there is no advantage, but much disadvantage, in deferring the operation. Such a course would mean the necessity of a second anesthetic, a harrowing period of suspense to the patient and her family, a greater economic outlay, and a theoretic possibility of dissemination of the growth by one mechanism or another. All these disadvantages are avoided by immediate operation, if other considerations indicate this. This plan of course presupposes that the surgeon is qualified to be his own pathologist, or else that the operating room is closely linked up with the laboratory, so that an immediate diagnosis is available.

SUMMARY AND CONCLUSIONS

The study of the material on which this paper is based, and also a review of the literature, indicate that the lymphatics constitute by far the most frequent route for the extension of corporeal cancer to the ovary. This is what we would expect from our knowledge of cancer characteristics in general. Some of the evidence for the spread of corporeal cancer by the lymphatics may be summarized as follows: (1) The lymphatics have been shown to be chiefly responsible for the spread of carcinoma elsewhere. (2) Our knowledge of the lymphatic drainage of the uterus explains quite satisfactorily the distribution of the metastases, in the ovary as well as elsewhere. (3) Emboli of cancer cells are often found in the lymphatics. (4) Cancer metastasis of the tube is often found either with or without ovarian metastasis. It not infrequently occurs in the wall of the tube, perhaps without mucous membrane involvement, as would be expected if implantation were important. (5) The surface of the ovary is characteristically smooth and uninvolved, as we would expect with lymphatic metastasis, but not with direct implantation of cancer particles on the

surface. (6) The bilateral distribution so common with ovarian carcinoma suggests a lymphatic source rather than one by implantation. (7) The lymphatic theory, rather than implantation, explains ovarian metastasis with pyloric cancer, although this problem has not yet been satisfactorily solved. (8) The finding of free cancer particles in the tube in cases of uterine cancer does not justify the conclusion that associated pelvic cancer is caused by implantation of such particles, even in the event of their being regurgitated through the tube. More often these particles are probably moving downward toward the uterus. (9) The cases reported by Sampson of supposed implantation cancer of the ovary are far more logically explained as due to lymphatic dissemination. (10) In view of the demonstrated importance of the lymphatics in the spread of carcinoma, we are not justified in explaining a case as due to direct implantation unless the lymphatic route has been excluded. So far as I know, this has not as yet been done in any case. (11) Of the 7 cases of ovarian metastasis herein reported, 6 appear to be logically explained on the lymphatic theory, while in the remaining one direct extension may have been the chief factor concerned. A study of cases reported in the literature bears out the impression that the lymphatics are the important route for dissemination.

In the operative removal of the cancerous uterus, cognizance should be taken of this prime importance of the lymphatics in the dissemination of cancer cells. The danger of squeezing such cells into the lymphatics is lessened by the early application of clamps to the broad ligaments, and by avoiding the use of strong compressing clamps to the uterus. The ligation of the fimbriated extremities of the tubes, as advocated by Sampson, is an extra precaution which seems worth while, especially where there is suspicion of tubal involvement.

Issue is taken with the advice of Sampson to avoid preliminary curettage except where there is no suspicion of cancer, or where the patient's condition contraindicates radical operation. Such a policy, if generally adopted, would inevitably lead to many unnecessary hysterectomies and a certain number of unnecessary deaths. The evidence as to the danger of diagnostic curettage in such cases is very inconclusive, while the evidence as to its indispensable value must be attested by every practicing gynecologist. The ideal plan, except in very obvious cases, is to curette, to make an immediate diagnosis from the frozen section, and to proceed at once with the radical operation if malignancy is found.

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TOTAL VERSUS SUBTOTAL ABDOMINAL HYSTERECTOMY

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THE free discussions at various medical meetings on the subject of total or subtotal hysterectomy in cases of benign lesions leave no doubt but that the opinions of the medical profession with regard to the respective merits of the two operations are greatly at variance. This is what might be expected since there is a legitimate field for each operation. During the last ten or fifteen years there have been advocates, among leading gynecologists, of the more general adoption of total hysterectomy for fibromyomas when removal of the uterus is indicated. However, these men have had a great deal more experience with this operation than most surgeons, and it is hardly fair to compare their results or their statistics with those of men who have not had the same training or experience.

The length of time involved in the performance of the operation has an important bearing on the results. Any operation that cannot be completed within an hour will be followed by more or less shock, which in itself predisposes to postoperative complications and high mortality. If total hysterectomy is performed only in simple cases and no attempt is made to remove the cervix at the primary operation, if there is inflammatory disease of the adnexa or degeneration in a low-lying fibroma, or if the patient is obese, the risk should be but little more than that of supracervical amputation. On the other hand, if total hysterectomy is performed only in the complicated and difficult cases, especially those in which there are complications as the result of inflammatory disease, the mortality and morbidity will be much higher.

Myomectomy should be performed during the child-bearing period in preference to either of the more radical procedures when it is practicable, but if tumors are multiple the former may be more difficult, and in some cases in which the cervix is in good condition it is advisable to remove the uterus by the supracervical method. If possible some of the endometrium above the level of the internal os should be saved in the hope of retaining menstrual flow. I am convinced that total hysterectomy should be performed by experienced surgeons in most cases in

which hysterectomy is advisable and the patient is in good general condition. In the more difficult cases it is advisable to remove the body of the uterus with its associated tumors first; then by grasping the cervix with a tenaculum forceps it can be much more easily removed secondarily. The subtotal operation, however, should be the operation performed by the occasional operator, and the cervix should be removed only if it contains a definite lesion. As the experience of the surgeon increases he will, as a matter of course, remove the cervix at the primary operation in more cases. I am quite sure that if this rule is followed the mortality will increase but slightly, if any, and morbidity will decrease. Kahn's* advice to examine the cervix carefully and curette

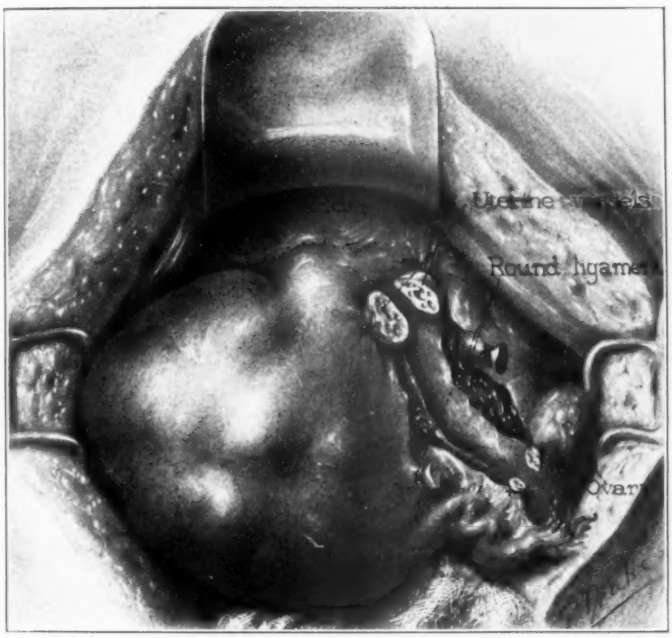

Fig. 1.—Round ligaments. Ovarian and uterine vessels tied.

the uterine cavity before the abdomen is opened in all cases in which hysterectomy is to be performed will help in the decision as to the extent of the operation necessary.

Many surgeons are inclined to regard removal of the body of the uterus with the fibromyomas attached as the important part of the operation, and thus to overlook other significant gynecologic considerations, such as the state of the lower pelvic diaphragm, and lesions in the cervix uteri. As a substitute for total hysterectomy, coning out the cervical canal has been advised. In this manner most of the cervical mucosa is removed and the tendency to leucorrhea greatly reduced, but if the

*Kahn's paper was listed but not read at the Meeting of the Western Surgical association, Duluth, Minnesota, October 14-16, 1926.

cervix is lacerated it is practically impossible to remove all the cervical mucosa without removing the entire cervix. The danger of malignancy is ever present in these cases. As a protection against infection, cutting the cervix with cautery and thorough destruction of the cervical mucosa by the free use of the cautery before the abdomen is opened for subtotal hysterectomy, as advised by Cashman, have distinct advantages. There is no doubt that the immediate sterilizing effect of the cautery is excellent and that by it all the mucosa of the cervical canal as well as that

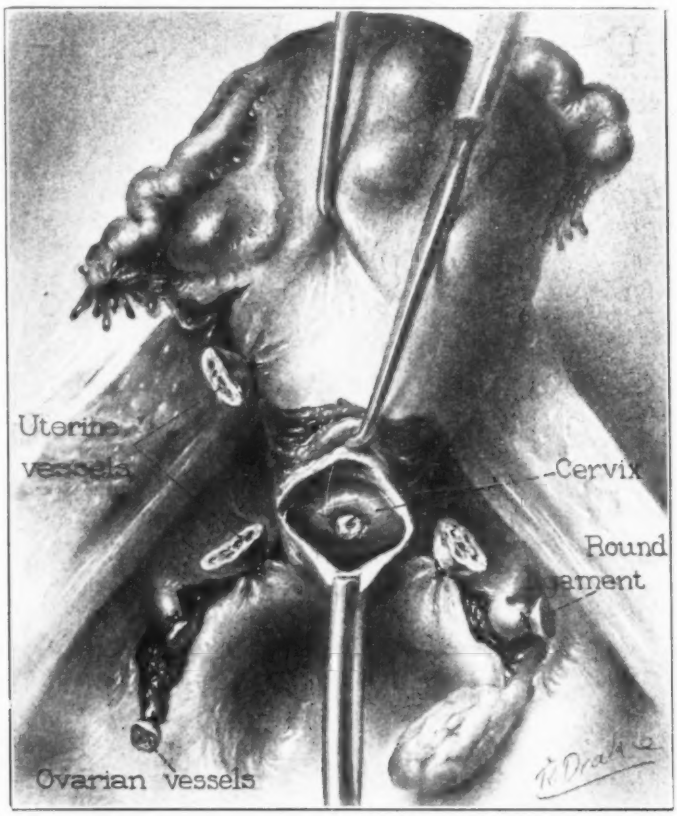

Fig. 2.—Opening vault of vagina posteriorly.

covering lacerations can be destroyed. However, the use of the cautery in any of these cases adds to the danger because the devitalized tissue is certain to become infected and increase the danger of peritonitis from being left close to, or in actual contact with, the peritoneum.

The mortality following either operation should be limited to accidental causes, pulmonary embolism being responsible for about 50 per cent of the deaths. The other most common causes of death are peritonitis, pneumonia, and nephritis. Thorough preparation of the vagina and cervix is a distinct protection against peritonitis. During the last

year there have been fewer cases of pulmonary embolism at the Clinic than usual. Such prophylactic measures as frequent change of position, and exercises, as advised by Wilson and Poole, breathing exercises, such as blowing up toy balloons, and the internal administration of thyroid extract, as advised by Walters and Coffey, are of real value. Care in the preparation of the patient and in the selection of the anesthetic will do a great deal toward preventing pulmonary and renal complications. I am convinced that ethylene administered by one experienced in its use adds to the safety of any operation. In cases of postoperative shock following extensive operations I have found the intravenous use of gum acacia solution, as advised by Keith and used by Dr. Ward and his associates extensively, of advantage and preferable to transfusion of whole blood, especially when the blood pressure is dropping.

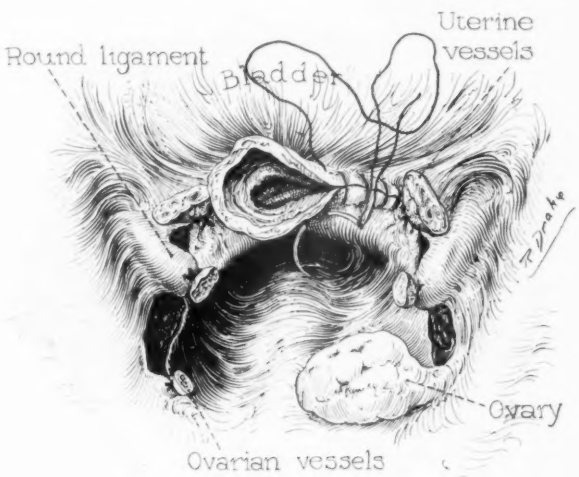

Fig. 3.—Closure of vault of vagina.

During the year 1926, total abdominal hysterectomy was performed in 229 cases in the Mayo Clinic, with three deaths, a mortality of 1.3 per cent. During the same year subtotal hysterectomy was performed 217 times with four deaths, a mortality of 1.8 per cent. Statistics vary a great deal, but in the hands of experienced surgeons the mortality for either operation should not be more than 2 per cent. Considering the fact that many of the patients are very anemic and others cachectic as the result of absorption from degenerating tumors or infection the mortality is surprisingly low and, therefore, hysterectomy may be regarded as one of the safest of abdominal operations.

During the last five years twenty-nine cases of carcinoma of the cervical stump have been observed in the Mayo Clinic. Twenty-one (72 per cent) of these occurred one year or more after the subtotal hysterectomy; 55 per cent occurred at least three years afterward; in one case there was an interval of fifteen years. Besides these there were forty-

five cases in which leucorrhea was troublesome; in some cases this began after removal of the supravaginal portion of the uterus. In many other cases there was a complaint of slight leucorrhea or cervicitis, and this was probably responsible for a certain amount of lumbosacral backache and bearing-down sensations, as the result of the spread of infection into the surrounding tissues, especially along the uterosacral ligaments. In such cases there is also the possibility that the cervicitis may be the focus of infection in other parts of the body, as pointed out by Moench, Curtis, Sturmdorf, Langstroth, and others.

Many of the objections to total removal of the uterus, such as the difficulty of the operation, shortening of the vagina, loss of support for the vault of the vagina, and added risk of peritonitis or infection and injury to the ureters, bladder, and sigmoid, are only relative. They depend in large measure on the surgeon's experience with this type of operation.

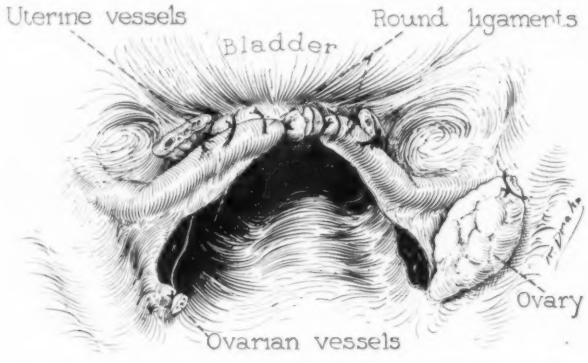

Fig. 4.—Fixation of round ligaments to vault of vagina.

Stitching the round ligaments, the uterosacral ligaments and the stump of the ligated uterine vessels firmly to the vault of the vagina, holds the latter in its normal position; there is no more tendency toward sagging or toward the development of a vaginal hernia than there would be if the cervix were left. Repair of the pelvic floor following either operation is just as important if there have been extensive lacerations of the perineal body. Important structures will not be injured if the normal anatomy is understood and care is taken in the application of forceps and in suturing to control bleeding.

Peritonitis should not occur more frequently from cutting across the vault of the vagina instead of the cervix. In fact, I believe there is little danger if one prepares the vagina with the patient in the lithotomy position after she is anesthetized, using a self-retaining weighted speculum and a tenaculum on the cervix, freely applying 3.5 per cent iodine, and plugging the cervix with a strip of gauze dipped in this solution. In case of purulent endometritis or a degenerating intrauterine submucous

fibromyoma, I always close the external os by suturing. Good exposure is obtained by a long low median-line incision, with the patient in the Trendelenburg position, a self-retaining retractor in place, and the intestines and omentum well packed off. I like to protect the intestines with a sheet of rubber dam (45 by 60 cm.) before inserting an abdominal pack.

TECHNIC OF ABDOMINAL HYSTERECTOMY

The round ligaments are first cut close to the horns of the uterus and the distal ends ligated. If the patient is past forty I usually remove both ovaries and tubes by cutting and ligating the infundibular ligaments with the ovarian vessels close to the brim of the pelvis. In younger patients I save the more normal-looking ovary but remove both tubes (Fig. 1). If both ovaries are badly infected I remove both but transplant the more normal-looking parts into the anterior abdominal wall. The peritoneum is then cut completely around the cervix and stripped back, in front with the bladder, and behind with the uterosacral ligaments. The uterine vessels are next

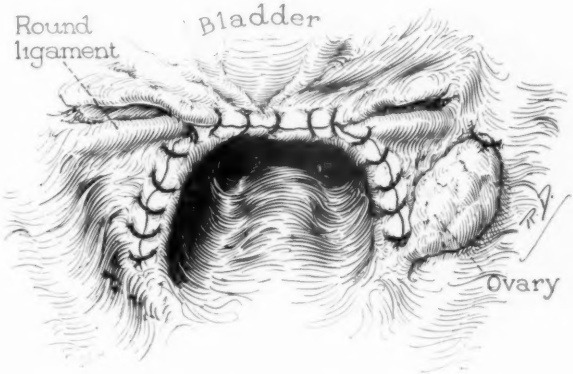

Fig. 5.—Covering of raw surface with peritoneum.

secured with three hemostats on each side just before they turn to follow the side of the uterus, then cut, two hemostats being left on the proximal side. If a subtotal operation is to be performed in an elderly patient, the cervix is somewhat coned out. If the patient is a young woman, a small amount of the body of the uterus is left, as well as the cervix. If total hysterectomy is decided on, traction is made on the body of the uterus and the cervix is separated from the bladder, the pelvosacral ligaments, and vault of the vagina by the use of the curved scissors (Fig. 2). I prefer to open the vagina posteriorly first and at once further sterilize it with 3.5 per cent iodine and leave a strip of gauze saturated with this solution in the vault. The incision is then carried around the cervix and the uterus is removed. A running mattress stitch is used to close the vault of the vagina (Fig. 3); the cut edge of the mucous membrane is turned down without stitching into it, and the closure reinforced with a second row of locked sutures. The uterine vessels are then tied en masse by a double suture. The cut ends of the round and uterosacral ligaments and the stumps of the uterine vessels are then firmly stitched to the vault of the vagina with chromic catgut and the raw surface completely covered with peritoneum (Figs. 4 and 5). This step is important. Probably the most serious complication as the result of any pelvic operation is the formation of adhesions from a loop of small

bowel low in the pelvis, thus predisposing to obstruction. There should not be any more raw surface after total hysterectomy than after subtotal hysterectomy, and there should not be any need of peritoneal drainage in any case.

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TRANSPLANTATION OF THE URETERS INTO THE BOWEL TO SECURE SPHINCTERIC URINARY CONTROL IN INCURABLE VESICOVAGINAL FISTULA

By Reuben Peterson, M.D., Ann Arbor, Michigan

Occasionally a vesicovaginal fistula is encountered which cannot be repaired by a plastic operation or a series of such operations. Such cases occur in women who have been terribly mistreated at their confinements. Much of the anterior wall of the bladder has been torn away by instruments or has sloughed away after their use. The bladder with a defective vesicovaginal septum is fixed in the pelvis by adhesions due to infection. Not infrequently it is impossible to locate the cervix amid the adhesions. The fixed uterus can be made out by abdominorectal examination, but the external os is not visible although present, as proved by the passage of menstrual blood.

Every failure to close such vesicovaginal fistulas makes the scar tissue denser and more rigid and increases the chances of failure at the next attempt at closure. The final result in such cases is perhaps to reduce the size of the fistula but to leave the patient dissatisfied and complaining because of the constant dribbling of urine.

Another and even worse variety of vesicovaginal fistula is where the vesical sphincter and adjacent portions of the urethra are absent. Such a condition can result from extensive obstetric trauma, subsequent infection and sloughing or from malignant disease of the urethra necessitating removal of the entire duct. Even where an urethra has been reconstructed, usually after a series of operations, the patient is dissatisfied since sphincteric bladder control can never be supplied once it is completely absent. With such a reconstructed urethra the bladder, after filling to a certain level, overflows, being especially troublesome when the woman is on her feet.

With such conditions present it is but natural for the surgeon to consider whether the urinary stream cannot be diverted into the lower bowel and the passage of urine controlled by the anal sphincter. Obviously this can be done in two ways, one by severing the ureters from the bladder and anastomosing them with the gut, the other by making an additional distulous opening connecting vagina and rectum and closing the introitus.

A review of the literature will show that both these methods were used on human beings as far back as 1851. In that year Simon transplanted both ureters into the rectum in a thirteen-year-old boy suffering from exstrophy of the bladder. The patient lived a year and died of diseased ureters and kidneys.

In the same year (1851) Maisonneuve, in the case of an incurable vesicovaginal fistula, made an opening between vagina and rectum and closed the introitus. The vaginorectal fistula could not be kept open and the patient died of septic phlebitis. The operation had been advocated in 1836 by Jabert and by Berard in 1845 but never executed.

In the seventy-five years which have elapsed since these first attempts, both types of operations have been employed in many cases with varying results. It is not my purpose at this time to review statistically such operations since that has been done at some length elsewhere. As far as possible I shall dwell upon the principles underlying methods of diverting the urinary stream into the lower bowel in order to determine, if that be possible, whether or not the gynecologist is justified in employing either of the two types of operations mentioned above in relieving patients with incurable vesicovaginal fistulas, who otherwise would remain in a deplorable and pitiable condition.

In 1900 I gave to the Society the results of experimental work on dogs in an article entitled *Anastomosis of the Ureters with the Intestine*. At that time I had no opportunity to operate upon human beings, and I doubt very much if I would have done so, feeling as I did about the dangers of ascending renal infection. As far as I could judge then from my experimental work and from a study of the literature, when severed ureters were implanted in the bowel, ascending renal infection always resulted, and inasmuch as it was impossible to determine in advance the extent of such infection I did not consider the operation justifiable. However, while the chance of ascending renal infection cannot be denied after transplantation into the bowel of the ureters with intact ureteral orifices, the infection is, as a rule, of such a type that the chances of the individual's overcoming it are good, hence such operations are justifiable.

These opinions governed my actions when in 1904 I made a vesicovaginorectal anastomosis with colpocleisis in a woman of sixty from

whom previously I had removed the entire urethra for carcinoma. This patient led a very comfortable existence for seven years, finally dying of a recurrence of the malignant disease in the cecum.

In 1916, a similar operation was performed upon a patient of thirty-eight, who had been left with an incurable vesicovaginal fistula following a frightfully mutilating obstetric operation. This patient made a good recovery, had perfect control over the urine with no apparent infection of the kidneys, at least, repeated examinations of the filtered urine removed from the rectum showed neither albumen, casts, nor pus. The last of such examinations was made within the past six months. This patient is well satisfied with her present condition of health eleven years after the operation.

There was only one primary death in forty-one cases of this operation collected by me and reported in an article published in *Surgery, Gynecology and Obstetrics* in 1917, and the functional results were excellent. The operation, however, is inapplicable to young women in the childbearing age, and in a number of instances it has been refused by women who have applied to the clinic for relief since it precluded copulation.

While I still believe that the experimental and clinical evidence is in favor of ascending renal infection where the cut ureter is implanted in the bowel, I think now that my former conclusions were incorrect to some extent. A great deal of the clinical evidence was based upon cases where the ureters and possibly the kidneys were infected prior to the transplantation. Infection of the ureters and kidneys was quite sure to follow the transplantation of such ureters. Experimentally, stricture of the ureter at its entrance to the gut was invariably followed by hydroureter and severe pyelonephritis. In extrophy of the bladder the ureters and renal pelves are damaged in a large proportion of cases. This may occur quite early in life. A majority of patients with exstrophy of the bladder die before the age of twenty from kidney infection.

In vesicovaginal fistula ureteral and renal infection is quite rare. In other words, patients with this defect are usually young and without infection of the urinary apparatus. Transplantation of ureters under such circumstances, provided such transplants be performed under certain well-known surgical principles applicable to ureteral work, will be followed by a minimum amount of kidney infection. The kidney infection may be so slight as to enable the organism to overcome it, provided the conditions of the transplant do not change. If the ureteral opening into the gut remains patent and not constricted with resulting hydroureter, ascending pyelonephritis will not occur to any great degree.

This is the probable explanation of the good results following transplantation of the vesical trigonum with its ureteral orifices into the

bowel (Maydls' operation), or the even more successful operation of Bergenhem who implanted extraperitoneally, the ureters with a rosette of bladder by separate openings into the rectum. These operations succeeded because the chances of ureteral constriction and resulting hydronephrosis were practically nil. The openings of implanted ureters were bathed in the septic fecal bowel contents but ascending pyelonephritis was reduced to a minimum since ureteral stricture was absent.

The same explanation accounts for the absence of pyelonephritis in the cloaca type of operation. While but little fecal matter passes through the rectovaginal fistula undoubtedly there is some contamination, yet pyelonephritis is practically absent after this operation. This is shown by my own case just quoted where the patient is living and well with no kidney infection after eleven years, and Keen's celebrated case where the patient remained in perfect health with no sign of renal infection twenty-two years after a similar operation. Sweet and Stewart experimentally showed that infection could not be traced along the mucosa of the ureter but was found in the lymph channels and that if the renal pelves were connected directly with the intestines no infection of the kidney occurred.

Hence, the problem of preventing renal infection in the transplantation of the severed ureters into the bowel resolves itself into the question of developing a technic which will produce the minimum constriction of the ureteral bowel orifice so that hydroureter will not result. The many attempts to form valves at the end of the implanted ureters have been futile except as they led to the implantation of unconstricted ureters. The indirect method of implanting the ureters in the bowel, that is, making a longitudinal incision in the intestinal wall through the serosa and muscular coats down to the mucosa and burying the ureter under these coats after drawing its end into the gut through a stab wound in the mucosa was fully described, and employed in 1899 by a Fellow of this Society, Dr. Franklin H. Martin. I employed this method extensively in the experimental work referred to above. Martin's idea was that the contraction of the muscular intestinal fibers would milk the ureters downward and prevent ascending infection. The method was a good one, not because the urine was milked downward but because it made possible the implantation with the least possible trauma to the ureter and less ureteral narrowing. In my own experiments I found "that the dog's ureter was extremely delicate and would not stand sutures passed through any of its coats without danger of resulting stricture. This was obviated by passing the sutures through its fatty and peritoneal envelope."

Coffey's operation of ureteral implantation is based upon the principles laid down by Martin, the burying of the ureters under the

serous and muscular coats of the intestine after securing the ureter by a ligature passed through its split end and drawn through a stab wound in the lower part of the mucosa. He obtains good results from his operations not because the implantation reverses what he calls static intraintestinal pressure, but because it is a technic which gives rise to little trauma and constriction of the ureter.

Credit, however, is due Coffey for popularizing the indirect method of transplanting the severed ureters into the bowel. Charles Mayo and others have employed the method extensively since the publication of Coffey's paper and the results of the transplantations under modern technic have been much more favorable so far as ascending infection is concerned.

Under such circumstances I felt justified in transplanting the severed ureters in the following two cases of incurable vesicovaginal fistula.

CASE 1.—Mrs. A. P. Admitted to the Obstetrical and Gynecological Clinic of the University of Michigan Hospital January 6, 1923. She had been unable to control her urine since a very severe operative delivery November 15, 1922 following which she was in bed four and a half weeks. Examination showed a very large vesicovaginal fistula with rigid walls. An unsuccessful attempt was made to close this fistula on January 18, 1923. She left the hospital unimproved and was operated upon a second time by a surgeon in Detroit, in 1925; this time by the abdominal route with no improvement.

The patient reentered the clinic January 27, 1926. Examination at this time showed a markedly contracted vagina beginning one and one-half inches above the introitus through which the forefinger could not be passed. The cervix was buried in a mass of cicatricial tissue and could neither be palpated nor exposed by the speculum. It was judged to be impossible to close the fistula by a plastic operation and ureteral transplantation was proposed and accepted by the patient who said she was willing to take any risk if she stood a fair chance of being made comfortable.

On February 18, 1926 with the assistance of Dr. Carl W. Eberbach to whom I am greatly indebted for aid in this and the other case to be reported, an extraperitoneal anastomosis of the right ureter with the sigmoid was performed with the following technic as briefly described as possible:

An incision was made from just above the symphysis upward nearly to the umbilicus and one inch and a half from the anterior superior iliac spine. The oblique muscles were severed by a long gridiron incision and the peritoneum exposed and pushed inward. The ureter was readily located, separated from the peritoneum, the latter incised longitudinally low down for two inches, the sigmoid located with intestinal forceps, brought into view through the peritoneal slit and secured in place by a few interrupted catgut sutures. The ureter was loosened down to the bladder, where it was clamped, the lower end being tied with catgut. An incision one and one half inches in length was made in the longitudinal band of the sigmoid through the serosa and muscles down to the mucosa. By careful dissection the incised intestinal wall was loosened from the mucosa one-half inch on either side. The end of the ureter was split for one quarter of an inch and the ureteral wall opposite the slit was perforated by a chromic catgut suture. A curved needle on each end of the suture was passed through a small incision in the lower part of the mucosa and through the gut wall one inch from the stab wound. The

ureter was then drawn into the gut slightly more than an inch and held firmly in place when the two ends of the suture were tied. The muscle and serosal coats were brought over the ureter and held in place by two layers of interrupted chromic Lembert catgut sutures. No sutures penetrated the wall of the ureter. A soft rubber drain was placed at the bottom of the wound which was closed in layers.

The patient made a good recovery; the wound healed well. She began passing urine per rectum in about thirty-six hours and was able to control it perfectly. Repeated examinations of the urine, drawn from the rectum and filtered, showed no albumin, casts, or signs of kidney infection.

The left ureter was transplanted into the sigmoid by a similar technic April 13, 1926, the patient in the interval having returned to her home. The wound healed well and convalescence was uninterrupted. She is able to retain her urine for four or five hours and does not have to get up at night.

She returned to the hospital for examination March 30, 1927, thirteen months after the last ureter was transplanted. She is pleased at the result of the operation since she is able to retain her urine longer than patients with normal bladders, at times as long as eight hours, and she does not get up at night. Her bowels move once a day. There are no signs of kidney infection. Pelvic examination reveals a vesicovaginal fistula an inch and a half above the introitus which admits the forefinger with difficulty. The cervix is buried in adhesions and cannot be felt or seen although the patient menstruates normally. She asks that something be done to the strictured vagina so that coitus may be rendered possible.

CASE 2.—E. B., aged twenty-three. Her trouble dates back to her confinement May 4, 1924 when, after having been in labor two hours she was taken to the operating room and the child turned and extracted. Since the childbirth she has been unable to control her urine. Her local physician operated upon her unsuccessfully, and she was sent to the University Hospital.

Examination showed almost complete absence of the anterior bladder wall and the entire urethra except for its posterior wall.

After some twelve plastic operations, including Ward's operation for the formation of an urethra, the vesicovaginal fistula was closed, but the new urethra was not a success. The newly formed meatus was constricted and there were two small openings at its base. She was much improved but rather unhappy since she had no control over the urine when on her feet and she had to earn her own living. She compared her condition with that of the patient whose case has just been reported who was in the hospital at the same time and begged that the same operation be performed upon her.

The right ureter was transplanted into the sigmoid November 15, 1926 with the same technic used with the first patient. The patient passed urine per rectum in about twelve hours and after a few days four ounces every four hours.

The left ureter was transplanted on December 26, 1926 by the same technic. Through some oversight the lower bowel and sigmoid had not been thoroughly emptied resulting in distension of the sigmoid and contamination of the wound when the stab wound was made through the mucosa. This resulted in seepage of urine and a fecal discharge from the wound for sometime. Eventually these disappeared and the patient was discharged from the hospital February 24, 1927 with perfect control over her urine. The latter showed no evidence of kidney infection.

In a letter received from the patient April 11, 1927 she says she passes her urine by the rectum about every four hours and at times gets up once at night. She is able to work and seems quite cheerful over her present condition when compared with her former miserable state.

COMMENT

I am convinced that the extraperitoneal is to be preferred to the intraabdominal route. No matter how careful the technic of the operation, something is liable to go wrong because it is not like ordinary intestinal surgery where accurate, tight approximation can and should be made. If the stab wound is too closely approximated to the ureter, hydroureter and hydronephrosis with ascending infection will result. This is illustrated by a recent case (to be reported in detail later) where just this condition resulted, necessitating removal of the right kidney.

If there be escape of urine or feces around the opening through the mucosa, the patient will succumb unless the operation has been performed extraperitoneally. Technically it is not more difficult since the ureters are readily exposed and easily transplanted.

I would not desire to expose the patient to the additional risk of transplanting both ureters at the same operation. When this is done you are burning your bridges behind you and something may go wrong.

What will happen to women who have had the ureters transplanted in case of pregnancy? Although not reported I am given to understand that one of Charles Mayo's cases went to full term after this operation and was delivered safely of twins. Evidently the dangers of pregnancy will depend upon how much if any renal infection may be present as a result of this operation. Obviously it would be unfair to these women to sterilize them for possible dangers which may never exist.

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TWO NEW IDEAS ON THE MECHANISM OF CERVICAL LACERATION DURING LABOR. A PRELIMINARY REPORT

By J. B. DeLee, M.D., Chicago, Ill.

OUR ignorance of the mechanism of cervical tears and of their frequency and extent is due largely to the teaching and practice of olden time. We were warned not to invade the parturient canal postpartum, unless there was hemorrhage. For years I have inspected the cervix after delivery and have been astonished at the amount of damage even natural labor causes and also amazed at the complicity of the injuries.

Fig. 1.—The usual laceration or lateral split of cervix.

It is generally taught that the mechanism of cervical laceration is very simple, that when the head distends the cervix it stretches as far as it can and then gives way at the sides, that is, in its congenitally weakest portions. The lacerations then would produce an anterior and posterior lip with a slit on each side like two longitudinal slashes in the cuff of a coat.

In a large proportion of the cases this is the mechanism, but in not a few instances two other mechanisms occur, and it is to these that I particularly wish to draw attention.

Fig. 1 shows the lateral splitting of the circularly overdistended cervix. The strong anterior and posterior lips have not been torn, but at each side, where the musculature and fibrous tissue are weaker, a split has occurred. The deeper tissues have retracted away from the torn surfaces leaving the external mucosa and internal mucosa approximated. In order to repair such a cervix properly it will be necessary to separate these two edges and dig down deep into the cervical body to fish out the retracted muscularis as shown in Fig. 2.

This form of laceration is the easiest to recognize and the easiest to sew up and is the one generally mentioned in the textbooks.

Fig. 2.—Detail of suture. Note how deeper tissues are lifted out to pass needle underneath.

In the second form of laceration, the mechanism of the tear is almost the same, but in this case while the musculature and fibrous tissues give way at the sides of the cervix, which are its congenitally weakest spots, the external and internal mucosae of the cervix do not give way and there is a submucosal parting of the tissues (Fig. 3). Inspection of such a cervix will show thick anterior and posterior lips with a very much stretched and excessively thin bridge of tissue on each side. By grasping the internal and external cervical mucosae with two tissue forceps it is usually possible to separate these two layers from one to one and a half

inches without any difficulty and discover in doing so that the very edges, apparently intact, in reality have been disunited. Occasionally, however, the edges are not torn at all, that is, the laceration is perfectly submucous (Fig. 3).

In most of such instances the observer would say there was no tear of the cervix since no damage is visible, but later on the cervix will gape and the lining mucosa roll out as in all deep tears.

The repair of such an injury is best made by splitting the mucosa and making a wound like that shown in Fig. 1, restoring the cervix as in

Fig. 3.—Submucous laceration of cervical tissues. Lateral fibers torn and stretched.

Fig. 2, digging out the deeper muscle and fibrous tissue, and lifting it up so as to pass the needle beneath it.

The third form of laceration of the cervix is much more complicated and not so easy to repair (Fig. 4).

The mechanism of the tear is complicated and hard to understand, but I believe it is as follows. The cervix is dilated radially to the utmost and the damage to the tissues is general, that is, all the fibers are stretched beyond their limit of endurance. The internal mucosa of the cervix become edematous and is ripped off its base, prolapsing through

the external os. The appearance of such a cervix after delivery is not unlike that of the everted anus of a horse (Fig. 5).

The repair of such a cervix is not easy and I have not tried it often enough to make a final recommendation. In a few cases I have attempted it as is shown in Fig. 6 and the results have been good. The edge of the cervix is pulled down with ring forceps while the mucosa is pushed up into the uterus with the four fingers of the left hand, the thumb making counterpressure on the outside. The vagina and bladder are held up by means of a suitable retractor and are not endangered. While the cervix is thus restored to its normal condition at the stage of

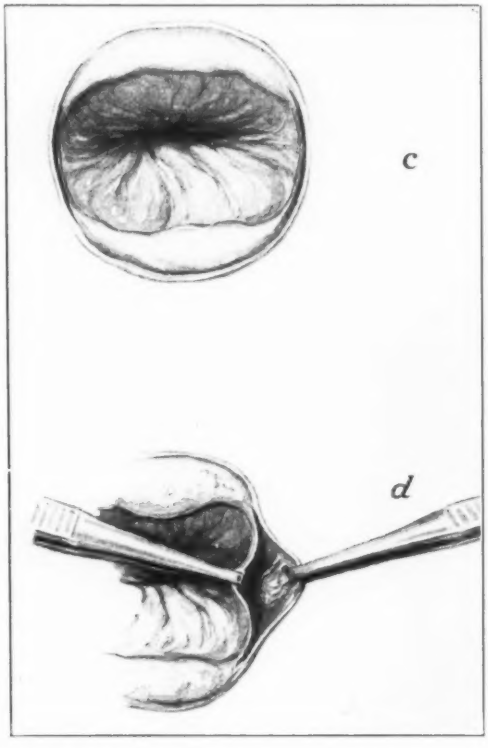

Fig. 4.—*c*, Same as Fig. 3. Shows edges of mucosa split. *d*, Edges of split mucosae separated to show separation of deeper structure.

full dilatation three sutures are placed at about the junction of the vagina with the cervix, operating from the vaginal side. These sutures go down into, but not through, the internal mucosa and they hold this prolapsed layer in place until healing is well under way. The procedure is repeated on the posterior lip if necessary but this is seldom. It is similar to the technic of the operation for prolapsus urethrae.

It is not rare to find combinations of these three mechanisms of cervical tear. The second and third forms of laceration are found together more frequently than with the first. The mechanism of labor has a great

deal to do with the formation of these tears but still more depends upon the congenital structure of the cervix and the presence of any kind of disease.

Cervices at birth vary very much in shape and quality. At puberty changes occur in this organ which changes, likewise, are variable and I have often thought that the cervix, as well as the uterus, is under some endocrinal influence. Acquired diseases produce still further variations in the cervix and all these have a great deal to do with the mechanism of dilatation during labor and with the mechanism of the formation of lacerations.

Fig. 5.—Thinning and overstretching of whole cervix with prolapsus of the endocervical mucosa.

I am at present making and collecting models of cervices during various periods of pregnancy, labor, and at the final examination before discharge after labor and I hope at no distant date to be able to report further on this subject.

With greater knowledge of the pathologic and surgical anatomy of cervical tears, our difficulties in their repair have increased. It has become a complicated technical operation to attempt to restore the cervix to its original condition. Under ether it is not as bloody a procedure as under ethylene, but in either case it takes much time, and exposes the patient to some slightly increased risk of infection. For these reasons,

the question may well be raised: is an extensive primary repair to be recommended to any but obstetric specialists? Or would it not be better to delay the repair until later in the puerperium as Dr. Hirst did, or Dr. Coffey, who takes all his puerperae to the operating room on the

Fig. 6.—Suggestion for the repair of the condition shown in Fig. 5. The protruding mucosa is pushed up and sewed in place by a few catgut sutures.

ninth day and does a typical Emmet trachelorrhaphy? Or should we let the woman go with only a partially restored cervix until the examination at the eighth week discloses its state, when we could cauterize it or sew it if necessary?

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THE AMNIOTIC FLUID AND ITS QUANTITATIVE VARIABILITY

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IN OUR eagerness to solve those questions of obstetrics that deal with the immediate practical needs of our patient, we are apt to forget that until some of the more fundamental problems dealing with the physiology and physiologic chemistry of childbearing have been solved, we cannot make much progress. A good example of one of these still unsolved questions is the source and nature of the amniotic fluid, and the causes of its quantitative variability. It is surprising how little time has been devoted by American obstetricians to this and similar subjects.

The evidence on which the present article is based consists of the study of thirty-two cases of hydramnion, and one case of oligohydramnion, observed in my private practice and by the Obstetrical Department of Washington University since 1916. In all but four of these thirty-three cases sections of the placenta were available for microscopic study. In one recent case of hydramnion a considerable quantity of the fluid was obtained for chemical analysis. Special study was made of the histologic changes in the amnion and compared with the findings of about one hundred normal cases. In subsequent studies we hope to report further on the chemical consistency of the amniotic fluid and histology of the amnion and chorion in cases of normal or excessive amount of fluid.

Histologic studies, beginning with the work of Bondi,[1] Mandl,[2] and Forsell,[3] have indicated that the amnion is more than a slippery covering for the sac in which the embryo lies. The amniotic epithelium, especially that covering the placental site, has been shown to be cylindrical, with characteristics pointing definitely to a secretory function. Fat globules in large numbers have been found filling the protoplasm of the cells and lying in pockets in the subjacent connective tissue. The unusual duplications of amniotic epithelium occasionally found were carefully described by Dr. Richard Paddock,[4] of our Department, in a recent publication. Paddock found in some cases several layers of a high columnar epithelium, and in others there was a squamous type of pavement epithelium with keratinization resembling that found in the skin. In none of the ten placentas in which these changes were noted was there any association with hydramnion or oligohydramnion.

BIOCHEMISTRY OF THE AMNION

Numerous experimental and clinical observations have demonstrated conclusively that the amnion is not a mere filter allowing certain substances to permeate from the maternal circulation into the amniotic cavity, but that it performs a definite metabolic function. The manner in which this is effected, according to Bondi, is that the amnion cell extrudes in the form of a vacuole, that material that it does not need for its own nutrition, and that this vacuole empties itself into the amniotic cavity. The fact that certain substances can pass directly into the amniotic cavity without entering the fetal circulation points definitely to a metabolic function of the amnion. Wislocki[5] has shown experimentally that substances like trypan blue injected into the amniotic cavity can pass into the maternal circulation, and can also enter the fetal circulation through swallowing of fluid by the fetus, or by passing directly through the amnion of the cord to the fetal vessels.

The direct passage of substances from the mother into the amniotic cavity without entering the fetal circulation is evidenced by Ottow's[6] case of sulphuric acid poisoning. Zuntz and Wiener[7] found similar conditions after the injection of sodium-indigo-sulphate. Juelich,[8] in a case of acute yellow atrophy, found the placenta very icteric, the amniotic fluid somewhat colored, but the skin of the child free from icterus. Holtermann,[9] experimenting with methylene blue, found that this substance in human beings passed very rapidly into the fetal circulation. It was not, however, eliminated by the fetal kidneys into the amniotic cavity. Instead of this a colorless leucobase of methylene blue was found in the amniotic fluid, presumably as a by-product of the amniotic epithelium.

Lycopodium powder, made very fine by previous treatment with ether, was injected into the eggs of a chick at various stages of gestation by Vrbitch.[10] He found the granules in the stomach and bowels within from one to thirty hours after injection. With rabbits also, where lycopodium was injected after laparotomy into the amniotic cavity and the uterus removed twenty-four hours later, the granules were regularly found within the stomach and intestines of the fetus. Vrbitch concludes that such swallowing of amniotic fluid is a regular process in the last half of the gestation period.

The biochemical composition of the amniotic fluid has been given considerable study in recent years. In amount the normal range is between 500 and 2000 c.c. It is a clear fluid in the earlier months, but cloudy from mixture with vernix and lanugo in the later months. It is composed of 98.5 per cent of water and 1.5 per cent of solids. These solids consist of albumins, fats, ferments, uric acid and its derivatives, and various salts. Reinlein and Heinlein[11] analyzed eighty-six

liters of amniotic fluid obtained from cows, making a special study for purin bases. They found benzoic acid, hippuric acid, and creatinine in small amounts; also histidin, methylguanidin, and a base isometric with tetanin. They conclude that the amniotic fluid contains a collection of the end-products of metabolism. Although these substances differ somewhat from those found in urine, the metabolic process seems very similar. Flössner and Kirstein[12] examined eight liters of human amniotic fluid obtained from normal cases without contamination, and found histidin, arginin, lysin, glycocol-betanin, sarcolactic acid, and aromatic oxyacids. Of special importance are the investigations of ferments in the amniotic fluid. These were carried on primarily by Polano[13] and his associates.[14] The presence of fat droplets in the amniotic epithelium and the tissues beneath it have long been noted. These are present in much larger amount in the free amnion than in that covering the placenta. Attention was directed by Polano to the presence of ferments in this portion of the amnion. In the amniotic covering of the umbilical cord these ferments are lacking. Of the proteolytic ferments he found a heterolytic, a peptic, a tryptic, and an autolytic ferment. Of ferments producing coagulation, he and his workers found chymosin and thrombase.

Interesting was the presence of a fat-splitting ferment, which, while it was found in both the placental amnion and the free amnion, was found in twice the quantity in the latter. This lipase, according to Kieffer,[15] has a definite function in the production of vernix caseosa. Recently, Maeda[16] found diastase in large amounts in the amniotic fluid and in the maternal blood, but very little diastase in the fetal blood. He concludes that diastase is produced primarily from the maternal blood. Lipase was found occasionally, and small amounts of pepsin, fibrin and chymosin, while trypsin was found in none of five cases examined. Polano considered the finding of these various ferments as conclusive evidence of the active functioning of the amniotic epithelium. In fact, he feels that the amnion may be regarded as a second line of defense between mother and child, serving to protect the latter from substances in the maternal circulation that may be toxic to so delicate an organism.

SOURCE OF THE AMNIOTIC FLUID

From the histopathology of the amnion, and these biochemical studies, there can be no reasonable doubt that the source of the amniotic fluid is the amnion itself, and that the theory held from the days of Hippocrates to the present century, that this fluid merely consists of fetal urine is unfounded. While it is true that in certain cases of absence of fluid, there is an absence of fetal kidneys, or a blocking of the fetal urinary passages, it is also true that normal amounts of fluid, and even hydramnion, have been found associated with these same

anomalies. That a slight admixture with fetal urine may occur shortly before or during labor seems probable, but this admixture is inconsiderable and has no functional significance.

HYDRAMNION (REPORT OF 32 CASES)

While the source of the fluid seems reasonably certain, the factors influencing its quantitative and qualitative production are practically unknown. Of greatest interest is the quantitative overproduction of this fluid, or hydramnion. We must, of course, realize that this may be due either to excessive production or to excessive retention of fluid. It would appear that normally there is an outflow as well as an inflow, and that, just as in the filling of a bathtub, the accumulation may result from a combination of an excessive inflow and restricted outflow, as well as from either factor alone.

The thirty-two cases of hydramnion recorded in this series occurred in about sixty-five hundred confinements at Barnes Hospital, from 1916 to 1926, making an incidence of about one in two hundred. The average age of the thirty-two mothers was exactly thirty years, and pluriparity was strikingly frequent. There were only five primiparas and twenty-seven multiparas, with an average of *five* children for each mother. The fact that normally, according to Lehn,[17] the average amount of amniotic fluid in multiparas is 1200 c.c. compared with 1000 c.c. in primiparas, would indicate that the relaxation of the abdominal walls and uterus in the pluriparous women tends mechanically to permit more readily the accumulation of fluid within the uterine cavity.

Pregnancy records of these mothers reveal few complications except those due to a crowding of the diaphragm by the accumulating fluid in the abdomen. In six instances labor had to be induced on account of the dyspnea thus occasioned. Three mothers showed a moderate amount of toxemia, and one had a definite diabetes. Of the twenty-three cases in which a Wassermann-Kahn test was made, only two showed evidences of syphilis. One mother had been tapped by her family physician for a supposed ovarian cyst, without apparent harm to her; although when she subsequently went into labor the child was stillborn and macerated.

Of complications of labor our series showed two instances of prolapsed cord, two transverse positions requiring version, and five cases of delayed labor requiring midforceps. In none was cesarean section indicated.

The children varied greatly in size. In spite of eight prematures, the average weight of the children was 3,350 grams, and eight of them weighed over 4,000 grams. In sex they were equally divided between boys and girls. Abnormalities of development consisted of six cases of anencephalus, and one child with syndactylism and a peculiar skinflap fixing the jaw down to the chest. This latter de-

formity doubtless interfered with any attempt to swallow, a point which will be later considered under the head of etiology.

In twenty-eight of the thirty-two cases sections of the placenta were studied. The careful saving and recording of this material by Dr. Otto Schwarz and his associates has made it possible to add important data, even though of a negative character to our knowledge of the placenta in hydramnion. Heretofore, such records have only rarely been available.

The most striking fact in our series was the large size of the placentas. They weighed on an average of 710 grams as compared with 500 to 550 grams for the normal placenta. In eight instances the placenta was unusually large weighing between 800 and 1100 grams. Abnormalities of length or insertion of the cord were not found. Of the various lesions outside of the amnion there was noted one case of a large angioma, one placenta circumvallata, two definite luetic placentas, two with cystic degeneration of the decidua, two with red infarcts, five with white infarcts, three with hematoma, one case of edema, and two cases with infection. Of these lesions the angioma could alone be held in a way responsible for the hydramnion.

Special attention was paid to the microscopic study of the amnion. These were compared with a large series associated with normal amniotic fluid. In only one-half of my cases, sixteen in all, was the amnion found in the sections; in the remainder it has been stripped off in imbedding or sectioning. These sixteen cases, however, showed no lesions that could with any definiteness be associated with the formation of the hydramnion. Twelve were entirely normal; of the remaining four, the lesions consisted of small areas of necrosis, areas of duplicated epithelium, and infection. Since these lesions were found in equal number in the group of amnions with normal amniotic fluid, no significance can be attached to them.

The amount of amniotic fluid in my cases varied between 2000 and 12,000 c.c. Of course, it is often difficult to measure the fluid accurately, but an approximation could usually be arrived at. There was but one case of typical acute hydramnion with sudden onset and extreme accumulation of fluid. Eight cases showed a moderate amount of fluid (2000-3000 c.c.), eighteen showed a large amount (3000-5000 c.c.), and six cases showed an extreme amount (over 5000 c.c.). In only one recent instance could a sufficient amount of amniotic fluid be obtained for chemical analysis. The history and findings were as follows:

Mrs. A. D. M. (private patient of Dr. McNalley), thirty-one years of age, five para, had had one tube and ovary removed two years previous to her present gestation. On account of persistent nausea and vomiting she entered Barnes Hospital in the eighth month of her pregnancy. Glucose was administered intravenously, but four days later she went into labor spontaneously. Four thousand cubic centi-

meters of amniotic fluid were present. An easy labor resulted in the expulsion of an anencephalic female monster weighing 1,750 grams. Seventeen hundred cubic centimeters of amniotic fluid were obtained uncontaminated for chemical analysis, and a small amount of fetal blood was also secured from the umbilical cord. An attempt to secure enough meningeal fluid for analysis was unsuccessful. The analysis of the amniotic fluid and fetal blood, made by Dr. Victor Gould, is shown in the accompanying table.

TABLE I

	HYDRAMNION FLUID	FETAL BLOOD
N. P. N.	14 mg. per 100 c.c.	33 mg. per 100 c.c.
Uric acid	10 mg. " " "	3.2 mg. " " "
Chlorides	700 mg. " " "	510 mg. " " "
Cholesterol	Trace	193 mg. " " "
	10 mg. " " "	
Sp. grav.	1.0063	
Ash	0.783%	
Total nitrogen	0.109 gr. per 100 c.c.	
Total protein	0.53 gr. " " "	
Total solids	1.565%	

These findings, as in the case of other investigators showed no material chemical difference between the fluid in hydramnion and in normal cases.

Attempting to analyze the etiology in our series of thirty-two cases, we were unable to find any clue at all in eight of them, 25 per cent. In the remaining twenty-four the following factors were tentatively held responsible:

6 Anencephalus
3 Twin pregnancies (two uniovular)
6 Large placentas
3 Unusually large children
1 Diabetes
2 Syphilis
1 Angioma of the placenta
2 Hypertension

GENERAL CONSIDERATIONS

While the type of hydramnion under consideration is that occurring toward the conclusion of pregnancy, it should be remembered that we may also meet with hydramnion in the early weeks of gestation. In a study of the cause of hematom-mole in 1902, I brought evidence confirming the hydramnion theory of Davidson.[18] These retained ovisacs showed a marked disproportion between the minute embryo and the large amniotic cavity, and in my view the accumulation of amniotic fluid or hydramnion, was a definite factor in the production of these peculiar blood moles.

A division into acute and chronic hydramnion has generally been made, but Marshall[19] calls attention to a third group that is not infrequent,—a chronic hydramnion with acute exacerbation. The typical case with tremendous accumulation of fluid in a short period of time is usually associated with uniovular twins. Experimental pro-

duction of hydramnion has been reported by Bruno Wolff.[20] He removed both kidneys from pregnant rabbits and found within two days thereafter a marked increase of amniotic fluid. This hydramnion was especially noticeable if the rabbits were near term. From chemical analysis of the fluid he concludes that the amniotic fluid in these cases is due to a compensatory secretion of urine by the fetus. Wolff does not believe that his experiments will explain all forms of hydramnion, but that where urine-forming substances are withheld in the blood, the fetal organs take on an added function to eliminate them.

Less convincing are the experimental efforts to produce absorption of amniotic fluid. Commandeur and Baussillon[21] injected intravenously 50 c.c. of a 3 per cent sodium chloride solution. After eight injections in a patient with hydramnion, over a period of three weeks, they noted a diminished circumference of the abdomen from 97 to 94 cm. Such slight changes may, of course, be due to other factors.

A clinical diagnostic point of some value in hydramnion has been noted by Pischzek and Schmidt,[22] who made studies of the capillary circulation in pregnant women. Since the capillaries of the nailbed are similar to those of the inner organs, they were not surprised to find in three cases of hydramnion a marked capillary dilatation.

Habitual hydramnion has been noted in a remarkable case by Luettger.[23] Syphilis was not a factor in this case. After three normal children there were eight pregnancies as follows: fourth, hydramnion, with child dying in one hour; fifth, dead, macerated child; sixth, normal living child; seventh, normal, but child died in four hours; eighth, slight hydramnion, stillborn at eight months; ninth, hydramnion, macerated child; tenth, hydramnion, macerated child; eleventh, hydramnion, macerated child. The placentas of the last two pregnancies were very large, weighing respectively, 970 and 1040 grams. No deformity of the child, nor heart or kidney lesion of the mother were noted. Floris[25] has also recorded two instances of repeated hydramnion in the same mother.

Of clinical importance is the fact that a considerable number of women with extreme hydramnion have been operated on or punctured in the belief that an ovarian cyst was present. This also occurred in one of our cases. Fortunately, it does not often produce bad results, although most women go into labor after such a puncture. Henkel,[24] who recommended abdominal puncture of the uterus to evacuate a large portion of the fluid, has found little support among obstetricians. The danger of bleeding from trauma of the placental site is considerable, and the benefits, as recorded by those who have tried this procedure, are very slight and transient.

Such radical measures are particularly to be deprecated when we consider the perilous future of these hydramnion children. Floris,[25] who reported 236 cases of hydramnion (many of them only slight),

found that while only 25 per cent of the children died at or before birth, 8 per cent died subsequently, but no mental or physical defects were found in these children later on. Of Poeck's[26] 136 hydramnion children at the Koenigsberg Clinic, fifty-six, or 40.4 per cent were found living and normal. He considers that the frequency of prematurity, faulty presentation and fetal monstrosities account for the immediate death rate, but that the ultimate prognosis of those that survive is good. Krahula[27] is very pessimistic about hydramnion children. Out of 72 cases he found 37.5 per cent with congenital deformities, and a primary mortality of 54 per cent. Of the remainder he found only 3 per cent living and normal after a period of time.

In my own series of thirty-two cases the incidence of deformities was 22 per cent, and the mortality, including two that died shortly postpartum, was 44 per cent.

ETIOLOGY OF HYDRAMNION

Hinselmann,[28] in Halban-Seitz's *Biologie u. Pathologie des Weibes*, gives a splendid summary of the various factors that may enter into the formation of hydramnion. Of first importance are deformities of the fetus. It would seem fairly certain that deformities of the fetus can be grouped into: (1) those that have no influence on the amount of amniotic fluid; (2) those that are associated with hydramnion or increased fluid, and (3) those that are associated with oligohydramnios or scanty fluid. Especially interesting is the incidence of anencephalus and those defects that interfere with swallowing, or that block the passage of fluid into the intestines, among the cases of hydramnion. Almost equally frequent is the appearance of oligohydramnios in children with defects of the urinary organs. Other forms, such as hydrocephalus, according to Lau,[29] are usually associated with normal amounts of amniotic fluid.

While there are exceptions in each group, the number of cases reported from various sources is so large that we must concede a definite relationship between the quantity of fluid and the type of deformity. This is particularly striking in the case of anencephalus, in whom it is reasonable to suppose that there is both an increased transudation of fluid from the meninges into the amniotic cavity, and an absence of swallowing of fluid by the fetus. Ample evidence has already been presented showing that such swallowing occurs normally in the latter half of pregnancy. The best evidence of this is the regular presence of lanugo hairs in the meconium of newborn infants. In controls that I recently made I found approximately from six to eight hairs in each cover-glass of meconium. In two of the anencephalic children in my series, however, where such an examination was made, not a single lanugo hair could be discovered, indicating that no swallowing had taken place. The absence of swallowing of amniotic fluid as a factor

in hydramnion is further emphasized by such cases of those of Theron[30] (stenosis of the esophagus); Buecheler[31] (struma blocking esophagus); Cramer[32] (micrognathia compressing trachea and esophagus); Voron,[33] Hempel[34] and others (aplasia of duodenum); Kleinman,[35] Lotz[36] and Schmidt[37] (five cases of stricture of esophagus). My own case of a fibrous band connecting jaw and chest should also be included in this group, although the findings could not be confirmed by autopsy. On the other hand, it is also true that occasionally normal amounts of amniotic fluid are found with stenosis of the small intestines, but Schmidt believes that in such cases enough fluid is absorbed by the stomach to permit an outflow by the fetal circulation.

Next in importance as an etiologic factor is twin pregnancy. Clauditz[38] found in forty-five cases of hydramnion, eleven instances of twin pregnancy, seven of which were uniovular. In 1876, Schatz[39] pointed out the tendency to polyhydramnion in one twin and oligohydramnion in the other. Randall[40] reports a case of uniovular triplets and hydramnion. In all reports of hydramnion from 10 to 20 per cent are associated with uniovular twins. In Benthin's[41] case the heart of the twin with excessive fluid was decidedly larger than the heart of the other; while the kidneys were of the same size in both. Apparently the hydramnion in these cases is due to circulatory conditions in the placenta not as yet clearly understood. In Goldschmidt's[42] case of acute hydramnion and uniovular twins he found areas of necrosis in the amnion epithelium which he held responsible for the condition. I doubt this interpretation since in one of the cases of uniovular twins in my series in which the amnion was preserved, no such necrosis was to be seen, the amnion being normal in all respects.

Forsell in his microscopic studies of the amnion in cases of hydramnion, lays special stress on the absence of a vesicular secretion in the amniotic epithelium in these cases. He argues that these vesicles are an indication of the outflow of fluid from the amniotic cavity to the mother via the amnion. When these cells were absent, this outflow could not occur and hydramnion developed. This speculation has not been supported by the observations of others and it is certain that in my sixteen hydramnion cases compared with a large number of normals, no such morphologic differences in the epithelium could be observed.

Unusual size of the children and placentas has been noted by many observers especially as a factor in the hydramnion of moderate degree. Fellner[43] found in a large series a direct proportion between the length of the child and the amount of amniotic fluid. This was a striking factor in my series. It is fair to assume that with unusually large placentas in lax-walled, multiparous women, the accumulation of a large amount of fluid might readily ensue.

Toxemic conditions of the mother form another group. In the presence of edema, hydramnion is not rare. Poeck found it a factor in 10 out of 136 cases. Syphilis may also be, to some degree, responsible for hydramnion, largely because it produces stasis in the placental circulation. In my own series, however, the incidence of syphilis was not more than we would naturally expect, nor have I found elsewhere definite proof of its etiologic relationship to hydramnion. The tendency to hydramnion in diabetes is much more definite. In the patient in my series unfortunately no examinations were made for sugar in the amniotic fluid or fetal urine. She had 4 per cent sugar three weeks antepartum and the amount of amniotic fluid collected was 2700 c.c. Offergeld[44] found dextrose in the fetal bladder urine at birth and attributes the hydramnion in part to fetal polyuria. Hinselmann[28] believes that the increased fluid in these cases is the physical result of the passage of sugar into the amniotic cavity corresponding to the tendency to edema in diabetes. He thinks that in the early stages suitable dietetic treatment may limit the development of hydramnion.

Finally, we must point out the association between hydramnion and circulatory conditions in the fetus and placenta. Alfandry[45] reports a case of malformation of the fetal heart, with anasarca and placental edema. More frequent is the association of angioma of the placenta with hydramnion. Hinselmann[28] gives a list of fifteen cases of angioma of the placenta associated with excess of amniotic fluid. He believes it due to the greater amount of the secretory surfaces in these cases. In the one case of angioma in my series of cases, an additional factor in the hydramnion may have been a deformity interfering with the swallowing of amniotic fluid.

OLIGOHYDRAMNION

Much rarer, but of equal interest, is the study of those cases with a deficiency or almost complete absence of fluid. The diagnosis will not be easy or certain unless the patient has been closely observed throughout her labor, but in the more extreme cases of oligohydramnion the clay-like consistency of the amniotic fluid, and the characteristic pressure deformities of the child leave no doubt as to the correctness of the diagnosis. A very striking instance of this condition came under my observation in 1920. The history was as follows:

Mrs. L. L., thirty-two years of age, married four years, was six weeks pregnant at the time of her first consultation, Dec. 3, 1920. She had had some slight irregular bleeding with cramps for two weeks previously, but under rest and codeine a miscarriage was averted. Confinement was due July 13, according to her menstrual history. During July the breech was down but not engaged in the pelvis.

The uterus was strikingly small and an attempt to correct the breech presentation was found impossible. There seemed to be little, if any, fluid in the uterus.

Fetal movements were felt only faintly. On August 7, twenty-four days after the expected date of confinement, labor was finally induced with repeated small doses of pituitrin. The breech entered the pelvis and complete dilatation was effected in sixteen hours. There was no bag of forewaters, and when the membranes were ruptured barely an ounce of fluid escaped. The fetal heart was good until the beginning of the second stage, but the child, a boy weighing 2800 grams, died during delivery which was not especially complicated. The child showed marked evidence of compression. Both ears were flattened out against the head, resembling the ears of an elephant. There was a pronounced curvature of the spine, and the lower extremities were curved in accordance with the shape of the uterus, with the feet markedly clubbed. In addition to this a moderate degree of hypospadias was present. Autopsy was not permitted, so that the question of internal anomalies of the urinary tract could not be determined. I have since confined this mother of two other children with normal amniotic fluid, and both children are living and well.

The placenta in this case was very small, measuring only 13 by 10 by 4 cm. The cord was 44 cm. long and attached 1 cm. from the margin. The maternal surface was unusually pale; one portion showed a hematoma 1 by 0.5 cm. in area. Numerous small infarcts were found on microscopic section. Of special interest were the changes in the amnion. This showed the epithelial layer broken into bits, with a few cells crowded together between flattened amorphous masses, that were probably remnants of necrotic epithelium. Over a considerable area, not over one-third of the amnion, cells were found intact. Whether this change in the amnion was primary and responsible for the absence of fluid, or whether it was secondary and due to pressure and scratching of the amniotic surface by the fetus, is difficult to say.

Vorlaender[46] found necrosis of the amnion in all three of his cases. Where any epithelium was found, it showed degenerative changes (vacuolization). He suggests that the permeability of the amnion after epithelial necrosis leads to a flow of fluid from the amniotic cavity to the maternal circulation.

In recent years attention has been called, especially by Neumann,[47] to the incidence of urinary deformities of the fetus with oligohydramnion. In forty-nine cases, it occurred nine times. Meyer-Ruegg[48] reports a case in which scantiness of fluid occurred in three successive pregnancies. Hürzeler[49] (3 cases) and Bertkau[51] found absence of kidneys in the fetus.

The occurrence of scanty amniotic fluid in cases of cholera has been noted and must be explained on the basis of an absorption of fluid by the mother from the amniotic cavity. According to Ahlfeld,[50] the absorption of amniotic fluid occurs normally in retained abortions, in twin pregnancy when one fetus has been compressed and ceased to develop, and in ectopic pregnancy that has gone to full term. Ahlfeld says we must assume either a primary absence of secretion of fluid or an absorption of fluid later in pregnancy. The absence of any amniotic bands or adhesions points, in his opinion, to the latter assumption. Further studies of the placenta and amnion in these cases will, in his opinion, help to solve this riddle.

The clinical characteristics of oligohydramnion may be summarized as:

1. Proportionately small size of pregnant uterus.

2. Greater frequency among primiparas.

3. Tendency to breech presentation, in which position the fetus adapts itself more closely to the shape of the partly filled uterus.

4. Prolonged pregnancy (from 20 to 30 days overdue).

5. Long, slow labor, with frequent necessity for manual or instrumental interference.

6. High fetal mortality during and after labor.

SUMMARY

1. Recent studies, especially biochemical analysis of membranes and amniotic fluid, point definitely to a metabolic function of the amnion.

2. While the source of the amniotic fluid is with reasonable certainty to be interpreted as the amnion epithelium, the causes of its quantitative variation in the form of hydramnion and oligohydramnion are still unknown.

3. Certain types of deformity in the fetus are associated with hydramnion, and other types with the absence of fluid. Malformations are very common in both groups.

4. No histologic changes in the amnion or chemical changes in the amniotic fluid in the cases of hydramnion have been found to explain its occurrence.

5. Necrosis of the amnion in cases of oligohydramnios is rather constant and may be an etiologic factor.

6. Unusually large placentas are found as a rule in hydramnion. Possibly the increased surface for secretion explains the increased fluid in these cases.

7. There is definite evidence of the physiologic swallowing of amniotic fluid by the fetus and the absorption of this fluid by the fetal intestines resulting in its return to the maternal circulation. When this outlet to absorption of amniotic fluid is blocked by some hindrance to deglutition or a stricture of the upper digestive tube, hydramnion results with striking frequency.

8. Twin pregnancy, more often uniovular twins, are often found, as a factor, especially in the acute forms of hydramnion.

9. Clinically, the prognosis for the child is bad in both groups; for the mother it usually means a complicated, but ordinarily not a dangerous labor. There is some tendency to repetition of the condition in the same person.

10. The predominance of primiparas in cases of oligohydramnion and of pluriparas in cases of polyhydramnion indicates that the physical resistance to expansion of the uterus by the abdominal muscles is of importance in the amount of fluid accumulation.

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TOXIC GOITER IN ITS RELATION TO THE GYNECOLOGIC PATIENT

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GOITER is frequently found in Michigan and its management must necessarily receive much attention from our surgeons. Especially is toxic goiter in its several clinical forms the subject of consideration, and as in other intricate fields, it is necessary that we spend much time and study on this particular phase of the subject in order to maintain a high standard of results and a low mortality. What we are pleased to call toxic goiter, however, is by no means confined to our section of the United States. Although perhaps less frequent, it is nevertheless a problem everywhere. In its less apparent and milder manifestations it is, I believe, frequently overlooked by those whose contact with the disease is small. A fairly large experience with it during the past twenty years, running parallel with a gynecologic practice has led me, therefore, to present this paper.

I shall consider the matter under several headings. What relation has toxic goiter to the functions of the pelvic organs and how far is toxic goiter caused or modified by gynecologic conditions? In the examination of the gynecologic patient, what symptoms or conditions should lead us to determine the presence or absence of the disease? With what other conditions is it apt to be confused and how may we distinguish them? When the presence of toxic goiter has been established, how may it affect our treatment of gynecologic conditions? What treatment of such a goiter should be advised by the gynecologist?

For the purpose of this paper I have included under the term "toxic goiter" all pathologic conditions of the thyroid gland which give rise to, or are definitely associated with certain familiar symptoms. I have made no particular attempt to consider separately its several clinical forms. As is already well known to you they are: a high metabolic rate, loss of weight, certain heart conditions associated with a rapid pulse, tremor, an enlargement of the thyroid gland, nervousness, exophthalmus, and certain vasomotor disturbances. They vary in constancy and appear in varying degrees. One or several may be absent. Each may well receive a considerable study to determine its significance in diagnosis, prognosis, and treatment. There are many cases in which the disease is so well covered, or mild, or its manifestations caused by other conditions, that without much study one may easily be led into error. There are some that in spite of it leave one in doubt.

I am not considering here pregnancy and its relation to toxic goiter, nor am I concerned with the ovary in its relation to the thyroid or other

endocrine glands. I am speaking only of toxic goiter. As far as the pelvic functions are concerned, we find no very close relationship. Women with toxic goiters occasionally have short periods of amenorrhea, otherwise their pelvic organs seem to behave about as usual. My observation would indicate that none of the various pathologic conditions found in the pelvis is influenced to any extent by toxic goiter, nor do such various pathologic conditions cause or influence toxic goiter. There seems to be no question but what certain infections have a part in the causation, but I have never found those of the pelvis acting in this capacity. We have made no extended investigation as to sex relations, but such patients do not complain to us in regard to these matters and we have hesitated to inquire . It is to be noted that but few of them become pregnant during the period of invalidism. In our experience also, toxic goiters do not occur oftener during the menopause than at other periods of adult life. It is rare in childhood, becomes frequent at about the age of twenty and continues to manifest itself up to an advanced age.

In the examination of his patient what symptoms or conditions should call the attention of the gynecologist to the possibility that she may be suffering from toxic goiter? First, a perceptible enlargement of the thyroid gland. Routinely, I think, every such patient should be examined for toxicity. If the patient has an adenomatous gland special attention should be directed to the heart for any evidence of myocardial degeneration. So commonly does such degeneration follow a gland of this type that surgeons are now commonly advising their removal in any woman over twenty-five years of age. An absence of apparent enlargement means little or nothing; toxic goiter frequently occurs without it. Again, every woman of whatever age who presents signs of myocardial weakness—fibrillation, dilatation, or both—is frequently suffering from toxic goiter. A loss of weight of say more than ten pounds, not otherwise easily accounted for, is another reason for suspecting this disease. There are few conditions that cause the great losses of weight that we meet with here (forty to sixty pounds is not extraordinary and twenty to thirty pounds is a common thing) though a slight or no loss is not a rarity. Nearly all toxic goiter patients have a good appetite and eat well, but lose weight in spite of it.

The rapid pulse which is so apt to be the first symptom to arouse our suspicious demands some little comment. In toxic goiter patients we should distinguish between the pulse rate under rest and under stimulation. A marked characteristic of the disease is that the pulse rate increases when the patient is stimulated. A slight degree of excitement, such as that produced by examination or the taking of the metabolism test, or the greater excitement, say of an approaching operation, increases the rate often to an extraordinary degree. It is a fair measure of the degree of increased excitability exhibited in this disease. Exercise and surgical operations also cause an increased rate. The pulse rate is

less important than the condition of the heart and is not a true measure of the seriousness of the case. As characteristic as the increased rate is the pounding, tumultuous action of the heart. It is very seldom met with in other conditions and only simulated by some of the heart lesions. In mild cases it may not be outspoken and in patients with rigid thoracic walls or emphysema it may be covered. As a symptom, however, it is one of the most constant and reliable signs that we have. A rapid heart is occasionally absent; one should not be deceived thereby. When, not suspecting any goiter toxicity, we perform a gynecologic or other operation, and obtain a pulse rate strikingly increased over what was expected, during operation or afterwards, we should suspect toxic goiter and search for other evidences of it. An increased systolic blood pressure with a low diostolic is characteristic of the disease. When combined with a rapid pulse it has distinct diagnostic value. Estimates of the systolic, diastolic, and pulse pressures are of even greater value as a guide in the conduction of the case.

Tremor in varying degrees is, as a rule, present. Its intensity is apt to vary with the degree of excitement. Usually the tremor is a fine one, but not always, especially in some of the more pronounced cases. It is most easily detected in the hands, though it usually extends over the body. It is a symptom which occasionally gives the clue to the diagnosis.

Vasomotor disturbances are flushing of the skin, especially of the head and neck, sweating, and a sense of unusual warmth. This sense of unusual warmth reminds one of that experienced during the menopause. As a rule, however, it is more uniform and less likely to come in waves. One of the most constant symptoms of toxic goiter is moist, warm hands. A mild thirst may be found, on questioning, to be present when vasomotor symptoms are prominent. They are very apt to be absent in the milder or atypical cases.

The nervousness which distinctly belongs to toxic goiter and is a part of the disease is of one type—a more or less constant feeling of tension and a desire to keep going. It reflects the increased excitability which is such an extremely important factor. It varies in intensity from that of the normal individual to great restlessness, mental excitement, and occasionally mania. The emotions are easily disturbed; women are occasionally depressed, but in the early stages, at least, these patients seldom remain so. Increased excitability may be the symptom to arrest the attention of the examiner. Increased fatigability and weakness are almost constant symptoms and cause much distress. In fact, they often bring the patient to the physician.

How may one differentiate toxic goiter from other conditions simulating it? Tuberculosis, so often associated with loss of weight and a rapid pulse should give us no trouble. A basal metabolism test settles the question in doubtful cases. The same may be said of pelvic infections of long standing. The so-called neurocirculatory asthenia may give rise to

some difficulty. There is increased fatigability and weakness. The emotions are easily disturbed. The anxiety and depression are not always entirely dissimilar. The pulse is rapid and the apex beat forcible; the tachycardia disappears during sleep which is not the rule with toxic goiter. The loss of weight is not excessive. The hands are more or less cold and clammy (an important diagnostic difference), and the metabolic rate is not disturbed. With care the two conditions should rarely give us any trouble.

Far greater difficulties are presented by the nervous patient whose heart action is easily disturbed, and who is emotional, has tremor under excitement, and is easily fatigued. Under rest the rate usually returns to 90 or less and distinctly neurotic symptoms are usually in evidence. A metabolism test is our chief dependence and with increased experience we are less apt to be in doubt, but our most difficult problems come in this class.

We not infrequently see patients who are distinctly neurotic and yet have mild or even a greater degree of goiter toxicity. We may often distinguish the manifestations of the one from the other. Patients that impress us as being overstimulated and who have one or more suggestive manifestations of toxic goiter but a metabolism test within the normal, we are apt to classify as "potential goiter." We have observed a few that later displayed distinct goiter toxicity. Eyes that are slightly staring should be included among the symptoms that suggest the disease.

The basal metabolism test is the best single aid that we have, and we use it in every doubtful case. In a general way an elevation of less than twenty is doubtful, and unless other symptoms are well pronounced we do not operate. With patients, however, who have adenomatous glands we are apt to advise operation if they present any signs whatsoever of toxicity. A bruit over the thyroid is pathognomonic of toxicity, but in my experience is not present in the great majority of cases. The same may be said of exophthalmus.

When the presence of goiter toxicity has been established, how may it affect our treatment of gynecologic conditions? It goes without saying that no gynecologic operation is going to affect toxic goiter favorably nor is the removal of tonsils or other foci of infection of any particular value. We have found them utterly undependable.

Unless the gynecologic condition is urgent, we should defer operation until the toxic goiter has been corrected and the patient returned to a nontoxic state or, at least, almost so. Plastic operations are the best examples of this. If the gynecologic operation is urgent, the mildest procedure possible to remove the crisis temporarily should be resorted to. A major pelvic operation in the presence of toxic goiter of even mild severity is made far more critical than it otherwise would be. If the gynecologic operation is very necessary, but can be deferred for a week

or more, the use of Lugol's solution (m. x, t. i. d.) with bed-rest may help to make the operation less critical.

How may we advise patients with toxic goiter? In the past prolonged bed-rest and sedatives was the most frequent method of treating such patients. At present it should be reserved for only the mildest and the doubtful cases. For all of the outspoken, the severe cases, and almost all of even the mildest, operation is by all means preferable. It is the most powerful means we have of controlling the disease and of placing the patient at once on the road to recovery. With greater experience in estimating the surgical limitations of a patient, with better technic, and the use of Lugol's solution, with bed-rest in the hospital, preliminary to operation it has become a comparatively safe procedure. A preliminary ligation is sometimes best even today, and the removal of the gland in two sittings may sometimes be done for the sake of safety. In the great majority of instances, however, a double lobectomy, leaving but little of the gland, may be performed. The mortality all told in operated cases compares favorably with that of our gynecologic cases and is very low. Patients with extreme restlessness and excitement and who do not improve under bed-rest and Lugol's solution and those who are maniacal are best not operated upon, but the percentage of such is small.

The use of Lugol's solution as a treatment for toxic goiter is not to be recommended. The improvement is of short duration and it removes one of the surgeon's chief reliances at the time of operation. It should be reserved for use before operation and perhaps afterwards. The use of the x-ray and radium is not advisable. The improvement, if it occurs at all, is slow, often only temporary, and the results are less certain than operation.

I do not wish to convey the impression that operation is ideal. Almost without exception patients do improve after operation. They regain their weight and their other symptoms improve. Most of them are completely restored to health within a few months or a year. Some of them, however, show signs of toxicity for a very long time and a few must necessarily be reoperated. Recurrences after years are unusual, but they do occur. One of the most remarkable results of operation is the restoration of badly damaged hearts, but a few seem to suffer a certain amount of permanent injury.

GRAND RAPIDS CLINIC.

THE TREATMENT OF PLACENTA PREVIA BY PROPHYLACTIC BLOOD TRANSFUSION AND CESAREAN SECTION

By Arthur H. Bill, M.D., Cleveland, Ohio

AS ONE glances over the recent literature of placenta previa, he must be impressed with the fact that while the most disastrous methods of treatment have been largely eliminated from practice, there has otherwise been very little change in the general attitude of the medical profession toward these cases. To be sure the practical elimination of the accouchement forcé has reduced the present day mortality considerably, but with the exception of the cases treated by this method the average maternal mortality of twenty years ago was as low as it is today, namely, 10 per cent or more in all cases and about 25 per cent in cases of central placenta previa alone. Placenta previa continues to be one of the most serious problems of obstetrics.

Death from placenta previa is usually due to postpartum hemorrhage. Seldom does death result in a case undelivered and in which no intrauterine manipulation has been performed.

There are two underlying factors in the etiology of postpartum hemorrhage in cases of placenta previa: (1) the condition of the patient at the time of delivery, and (2) the method of delivery.

The problem of the treatment of placenta previa is about as clear cut as any problem in obstetrics. It is solely one of hemorrhage and the treatment resolves itself into, (1) the replacing of lost blood to the patient, and (2) delivering the patient in such a manner as will cause the least loss of blood during delivery and the least danger of postpartum hemorrhage.

In practically all discussions of the treatment of placenta previa, attention is given solely to the relative merits of the numerous operative procedures which have from time to time become popular, the accouchement force, Braxton-Hicks version, bag, placental forceps, vaginal hysterotomy and abdominal cesarean section. Too little consideration is given to the fact that the amount of blood which has been lost by the patient previous to delivery is quite as important as the method of delivery and has a very definite bearing on the ability of the patient to withstand the delivery and on the problem of postpartum hemorrhage which is in most cases the cause of death.

In this connection let me emphasize the fact that in cases of postpartum hemorrhage, a definite vicious circle may be established inasmuch as the loss of blood may be sufficient to cause atony of the uterus, while the atony causes further hemorrhage. The atony of the

uterus may even be such that uterine packing will have little effect in controlling bleeding. Such a uterus will not respond to drug stimulation; in fact, the only hope of breaking the vicious circle thus established lies in blood transfusion. The effect of blood transfusion in producing uterine contraction is very remarkable. However, when the patient is in such a state of shock that the uterine muscle has entirely lost its contractile power, the question of restoring this function is sometimes problematical. No one wishes to be confronted with such a problem. The longer we practice the more surely must we be convinced that the only dependable treatment of postpartum hemorrhage is that of prevention.

There is no doubt whether that in the past there has been too great haste in delivering patients with placenta previa. Physicians have been so impressed with the idea that in the presence of antepartum hemorrhage the uterus must be emptied that they too often resort to a method of delivery when the patient has no chance whatever of surviving it. No attempt is made to determine whether or not an additional loss of several hundred cubic centimeters of blood, which must surely take place in any method of emptying the uterus, will be sufficient to send the patient into shock, or if you wish, establish the vicious circle previously mentioned. There must be greater deliberation to meet the requirements of the case in hand.

We would urge the more general use of the prophylactic blood transfusion, that is, transfusion given previous to the delivery so that the danger of postpartum hemorrhage from atony of the uterus may be avoided. Our experience has shown conclusively that if sufficient blood is given by transfusion before the delivery the type of postpartum hemorrhage just described will seldom occur.

Inasmuch as the patient may continue to lose blood it is of course important that there shall be no prolonged interval of time between the blood transfusion and the delivery. The ideal manner of carrying out this procedure is to have two teams in action, one for the transfusion and one for the delivery. While in most cases the two procedures are performed almost simultaneously it is our rule not to commence the delivery until the greater part of the blood has been given and in the most serious cases we take time to estimate the patient's condition after the first transfusion and thus determine whether or not she will require a second transfusion before the delivery. Estimation of the patient's ability to withstand the delivery is based upon the history and observation of loss of blood, the general appearance and apparent condition of the patient, and above all the blood pressure and the red blood count. No patient is delivered who has a red count below three million and a blood pressure below 90 to 100 systolic and 60 diastolic, until blood transfusion has been given.

Blood transfusion in obstetric practice is largely an emergency procedure. Haste is usually required and therefore such accuracy as will insure against the slightest delay which may arise from errors in technic. We, therefore, consider this procedure to be one highly specialized and worthy of the services of one member of the staff who has devoted much time to the perfection of details, not only of the technic of the transfusion itself but of blood reactions. Thus at the Cleveland Maternity Hospital, blood transfusion is under the direction of Dr. W. R. Barney, whose painstaking study of the possibilities and also the dangers of transfusion, and whose perfection of the technic have been of inestimable value in the development and success of our present policy.

In the delivery of cases of placenta previa there is a very noticeable tendency to adhere to the so-called conservative methods. By the conservative method is invariably meant delivery through the pelvis with the aid of the bag. This method of delivery with its slow dilatation is undoubtedly more conservative than the accouchement force, but has it stood the test? Is it really conservative? It certainly has not been the means of reducing the mortality to a degree which is at all gratifying. To be sure, in many cases in which the bag has been used the mortality was due to the fact that blood transfusion was not performed. However, it may be said that in some cases the fatality could be attributed directly to the method of delivery. Deep tears in the cervix may be, but unfortunately are not always, avoided by this method, but whether the cervix be torn or not, the placental site of the uterus is stretched and traumatized to some extent.

If the placenta is situated in the lower segment and over the os, there is enough muscular tone in that segment to control bleeding if the os remains closed and there is no trauma of the placental site. Evidence of this is seen in the clinical fact that after cesarean section for placenta previa there is no additional danger of postpartum hemorrhage when the uterus contracts well. However, in many cases in which the os has been dilated and the area of placental attachment has been stretched or torn, the insufficiently contractile lower segment of the uterus is unable to cope with the additional hazard and there is postpartum bleeding even though the fundus seems to be firm and in some cases even though there is no apparent tear in the cervix. (The condition seems somewhat similar to that which has been described as a paralysis of the placental site.) There is the greatest degree of postpartum safety in those cases in which the lower uterine segment has been left undistended and there has been practically no dilatation of the os.

Some years ago my attention was called to this fact by a case which is not in the list of cases reported with this paper. I shall mention it briefly.

Marginal placenta previa. Voorhees bag inserted, controlled bleeding perfectly and dilated the cervix. Fetal head followed the bag into the cervix and also controlled bleeding until it had passed through the cervix. Low medium forceps delivery. Cervix inspected and no tear observed. Postpartum hemorrhage. Uterus and vagina packed with gauze. Subcutaneously packing did not control hemorrhage. Saline given also intravenous saline with adrenalin. Preparation made for blood transfusion. Patient died before transfusion could be given. Cause of death was undoubtedly bleeding from the placental site.

In the article on placenta previa by Brodhead and Langrock in the January 1927, number of *Surgery, Gynecology and Obstetrics*, I noted two similar cases in the list of fatalities which I quote, as follows:

CASE 7.—A multipara at term with central placenta previa was treated by a de Ribes bag and version. Because of the parity of the patient, the use of the bag followed by version was the conservative plan of treatment. This patient bled to death because of continued hemorrhage from the placental site, which might have occurred even though cesarean section had been the procedure chosen. At autopsy no cervical laceration or uterine rupture was found.

CASE 9.—An viii-para at term with central placenta previa was treated by version. The case was unfavorable in that she had bled profusely before admission to the hospital. Although a conservative podalic version was done, there being no further hemorrhage during delivery, the patient died of continued bleeding from the placental site. A postmortem revealed an intact cervix and uterus.

These cases are, I think, typical of what may happen when a method of delivery is used which requires dilatation of the cervix. To be sure, this sort of hemorrhage is seen in only a small percentage of the cases so delivered, but certainly occurs in enough of them to account for a considerable number of the deaths which are reported. It is not seen in cases delivered by cesarean section. Why then do we continue to use the more complicated and more treacherous methods of delivery through the pelvis instead of the more conservative abdominal cesarean section?

If one were to analyze critically any list of fatalities from placenta previa, he could write across the histories of a large number of them the words, "Should not have been delivered without blood transfusion." Across the histories of nearly all the rest he could write, "Should have been delivered by cesarean section."

The accompanying table is a summary of the cases of placenta previa treated at the Cleveland Maternity Hospital.

I have divided them into two series, one previous to January 1, 1922, and the other from January 1, 1922, to the present time, because, during the time covered by the latter series, there was a somewhat more definite practice of making use of the prophylactic blood transfusion in cases of severe hemorrhage. While simultaneous blood transfusion and cesarean section were first performed by us in 1912, in the latter series a more careful estimation was made of the patient's condition to determine the advisability of transfusion. During this

SERIES A. CASES PREVIOUS TO 1922

45 cases delivered	Central placenta previa		23	
	Marginal placenta previa		22	
Method of delivery	Cesarean section		27	
	Bag and podalic version		17	
	Forceps		1	
Maternal deaths 5 or 11.1 per cent	1. On 13th day. Intestinal obstruction (?)			
	2. Hemorrhage, no transfusion			
	3. Hemorrhage, no transfusion			
	4. Hemorrhage, transfusion after cesarean section			
	5. Hemorrhage, 3 postpartum transfusions			
Fetal mortality	Stillborn	8	17 or	37.3%
	Died	9		

SERIES B. CASES SUBSEQUENT TO 1921

56 cases delivered	Complete placenta previa		25	
	Partial placenta previa		10	
	Marginal placenta previa		21	
Method of delivery	Cesarean section		40	Cases
	Podalic version		11	"
	Forceps		4	"
	Spontaneous		1	"
Prophylactic blood transfusion and cesarean section			12	"
Maternal death		1 or	1.78%	
Fetal mortality	Stillborn	10	18 or	32.1 %
	Died	8		
Cases requiring blood transfusion			12.4 %	
Cases delivered by cesarean section			71.4 %	

period, therefore, transfusion was not used as a measure of last resort but as a means of precaution where there was the least doubt as to the patient's ability to survive delivery.

In Series "A" one of the maternal deaths was due to an abdominal complication which possibly could not have been avoided. The other four deaths, however, were due to postpartum hemorrhages and could very likely have been prevented in view of our subsequent policy of prophylactic blood transfusion. In Cases 2 and 3 no blood transfusion was given, while in Cases 4 and 5 blood transfusion was performed after the delivery when there was found to be profuse postpartum bleeding on account of atony of the uterus.

Undoubtedly there was too great haste in delivering these patients without preliminary blood transfusion and the loss of blood caused by the delivery so depressed the patient that the uterus entirely lost its contractile power; so much so that even blood transfusion did not restore it. These cases show that the patient needs the greatest stimulation from transfusion at the time when the uterus is emptied; that there may be an immediate contraction of the uterine muscle which will prevent further loss of blood. They also illustrate the futility of blood transfusion in some cases when given after the patient is much exsanguinated.

Series "B" represents the results of our present plan of treating placenta previa. The first cesarean section for placenta previa which I performed was at the Cleveland Maternity Hospital in 1907. The

contrast between this method of delivery and the methods which I had previously used, namely, the accouchement force and later the Voorshees bag, was so striking that I published an article in the *Cleveland Medical Journal* advocating cesarean section as the best method of delivery in cases of central placenta previa and well-marked partial placenta previa. There were very few cases in the literature at that time and of course my argument in favor of cesarean section was not based upon any statistics of my own but upon the underlying principles involved. For some years afterward we performed cesarean section in the cases of complete placenta previa and in those in which the placenta overlapped a considerable part of the os, while the cases of lesser degree were treated by what we then erroneously thought to be the more conservative method of delivery through the pelvis. A realization of the danger of uncontrollable postpartum hemorrhage due to disturbance of the placental site even though there was no apparent tear in the cervix, as before discussed, gradually led to a more definite policy of performing cesarean section as a procedure of choice and really as a conservative procedure. Thus, in all cases of placenta previa whether central or marginal in which the cervix is practically undilated, cesarean section is now performed. This plan is based largely on the principle that the closed os and undisturbed placental site are most important factors in the prevention of postpartum hemorrhage in such cases.

The smaller number of cases in which cesarean section is not performed consists of those of lateral placenta previa in which the patient has been in labor, the os is considerably dilated, and the fetal head has descended sufficiently to control the hemorrhage by pressure.

No vaginal examinations are made in cases of antepartum hemorrhage. The diagnosis is based upon the history, the apparent bleeding, abdominal palpation and auscultation, and rectal examination. The elimination of vaginal examination and manipulation undoubtedly has been a most important factor in the reduction of maternal mortality. It is of course apparent that the differential diagnosis between central and marginal placenta previa is often not made before, but during operation. However, when cesarean section is performed it makes no difference which variety is present, the result being equally good in each. Postpartum hemorrage is rarely encountered when the condition of the patient is suitable for delivery, that is, when she has retained or has been given sufficient blood to insure uterine contraction. The uterus is not packed after cesarean section.

Prophylactic blood transfusion seemed to be indicated in about one-fifth of the cases. These naturally include the most serious cases. One typical case will serve to illustrate the possibilities of this method.

A multipara was brought to the hospital after a very profuse antepartum hemorrhage. Radial pulse could not be felt. Blood pressure could not be recorded. Blood count showed 1,500,000 red cells. Extreme pallor and air hunger. Transfusion of 500 c.c. of blood was immediately given. A fair radial pulse could then be felt. Blood pressure 80 systolic, while diastolic pressure was too low to be recorded. Red blood count 2,400,000. Patient still unfit for delivery. A second transfusion of 500 c.c. of blood was given and cesarean section performed just as the transfusion was completed. After the second transfusion the red blood count was 3,100,000, and the systolic blood pressure was over 100. There was no postpartum hemorrhage and the patient made an uncomplicated recovery.

The single death occurred late in the series, there having been fifty-three consecutive cases with no mortality. This case furnished more than a surgical problem. The husband of the patient absolutely refused permission to deliver or transfuse. He would not even allow typing of himself for use as a donor. Only after several hours of arguing and pleading and finally the demand of a priest, was permission given. The patient was practically moribund at that time. Two transfusions amounting to 1200 c.c. of blood were given and the patient was delivered by cesarean section. For one hour after delivery her condition seemed satisfactory with no evidence of hemorrhage. Then there was a sudden relaxation of the uterus with uncontrollable hemorrhage and death before a third transfusion could be given. It is evident that this patient could have been saved had delivery been performed when preparation was first made, five hours before the actual delivery. Two procedures could be suggested which might have prevented death even after the long delay. First in a case in which the patient is so nearly moribund before the transfusion it might be well to ligate the uterine arteries at the time of delivery. Second, in any case of postpartum hemorrhage, or a case in which there has been severe antepartum hemorrhage, the blood pressure should be taken postpartum at least every five minutes until the danger of hemorrhage is definitely passed, so that if there is a falling pressure another blood transfusion can be given before it is too late. A falling blood pressure is often the first evidence of bleeding during this period.

In conclusion emphasis should be laid upon the fact that while cesarean section is our method of choice in delivering cases of placenta previa, no method of delivery is safe in the more serious cases without prophylactic blood transfusion.

OSBORN BUILDING.

SURGERY VERSUS RADIOTHERAPY IN THE TREATMENT OF TUMORS OF THE UTERUS

By G. Brown Miller, M.D., F.A.C.S., Washington, D. C.

IT SEEMS to me that the time has come when gynecologists should have fairly well-defined views concerning the employment of surgery or radiotherapy in the treatment of fibroid and malignant tumors of the uterus.

To form an intelligent consideration of the relative indications and contraindications of the two methods of treatment we must bear in mind several facts. Some of these are the following: surgery has stood the test of time to a much greater degree than has radiotherapy. We know by experience that as time goes on the value of a supposed cure of disease as a rule, decreases, and its harmful action on the human organism is found to be greater than was thought. One has only to recall the various operations in surgery or the advent of certain drugs to have this fact brought vividly to his recollection. We hail a supposed cure with enthusiasm, expect the impossible, gradually grow skeptical of its beneficial effect, grow more suspicious of its harmful action, and frequently, finally discard it as worse than useless.

Surgery has gone through this trial and we have today quite definite knowledge of what it can accomplish, as well as to the objections to its use in the treatment of these tumors. In the case of radiotherapy there is quite a different picture. Its accomplishments for good or for evil are not nearly so well defined. There is no reason to think that radium or x-ray has any selective action on the cells of fibroid tumors or carcinomas. Kayserling and others have shown that the action of radium upon normal cells is exactly the same as it is upon carcinoma cells. The difference in its effects upon various tissue depends upon the stability, as it were, of the tissue. Its effects in the treatment of fibroid tumors of the uterus depends chiefly upon its action on the ovary by destroying its function and producing the menopause. Geller showed in rabbits that the so-called stimulation of small doses of radium, etc., for bleeding at the establishment of puberty instead of stimulating the ovary caused degeneration of this organ. In cancer the conclusions of Martin ten years ago are still logical, i.e., that there is no proof that any form of radio activity has any selective action on cancer or any other cells. The only difference is that cancer cells are more easily destroyed than most normal tissue cells. There is a maximum intensity of radiation which, when it is

just reached, cancer cells are destroyed, and connective-tissue cells in a measure are injured, while normal epithelium of the uterus is not injured. If this intensity is exceeded all the normal tissue in the neighborhood is injured or destroyed, while cells at a considerable distance may be stimulated to increased growth. (This last statement has apparently been demonstrated by several German observers, but it is questionable whether or not it is true.) We have never had a clear-cut conception as to what dosage is best, how long we must apply the agent, how long the interval should be between the applications, and what kind of application least injures the neighboring tissues. While this view will be modified as our experience grows, the application of radiotherapy is, and will probably remain, to a large degree empiric. The intensity of action of radium or x-ray varies inversely as the square of the distance. In trying to reach a deep-lying tumor the intermediate tissue is bound to be affected by it, and tumor cells at a great distance may possibly be stimulated to more active growth. We must remember also that intensive radium or x-ray applications cause changes in the blood (decrease in the red cells and hemoglobin and a temporary leucocytosis). That the mucosa of the small intestine is very susceptible to the action of these agents has been demonstrated by Warren and Whipple, Yugenberg, and others. Warren and Whipple state: "There can be little doubt that the small intestine can be injured by roentgen-ray therapy in human beings, and it is certain that such injury is serious." "We know," they say, "that roentgen-ray skin burns or ulcers are very chronic and we are able to state without reservation that the intestine can be so injured by the roentgen ray as to produce ulcers that are no less chronic than the familiar skin lesions." Yugenberg and others have shown that while the stomach is not very susceptible to the action of x-ray, that the small intestines are markedly so, comparatively small doses producing ulceration. The vitality of tissues is affected by x-ray and radium, produced probably by injury of the endothelium of the blood vessels, and when preoperative applications of these agents are made there may be interference with the healing of wounds. We are all familiar with x-ray and radium burns and sloughs. One of my patients who was given intensive x-ray treatment for a large fibroid tumor had at operation the most vascular and extensive adhesions of the intestines and omentum to the tumor; the operation was followed by sloughing of the skin and fat of the abdomen, and she developed a vesicovaginal and rectovaginal fistula. I think it beyond question that these untoward results were due to the intensive x-ray treatment prior to operation. We frequently see marked nausea and diarrhea following radiotherapy, and bloody stools and death have apparently resulted occasionally from its application over the abdomen.

Bagg and Little (*American Journal of Anatomy*, March, 1924) confirmed the observation of others that healthy mice known for generations to produce healthy offspring, when subjected to irradiation before mating, frequently produced abnormal offspring and that when the offspring of irradiated mice are bred together there may appear abnormalities in subsequent generations which by properly selected breeding may involve all the offspring. Healy says that experimental data upon the lower animals have shown that when the sex glands are sufficiently irradiated before fertilization that the following are aypical fetal reactions: (1) disturbed, abnormal, arrested development resulting in the formation of monsters, etc.; (2) a marked tendency to a progressive loss of fertility, and (3) a specific modification of hereditary mechanism and the production of inherited defects in the young, especially in the eyes. Irradiation during pregnancy produces disturbed, arrested, abnormal development with death of the embryos, absorption or abortion, stunting in growth, cataract, sterility, lesions of the central nervous system and blood vascular disturbances in the embryos.

A number of cases in human beings have been reported showing that similar results can be expected with them. An editorial in the *Journal of the American Medical Association* says, "It would seem on the basis of existing returns that the actual danger of developmental defects from irradiation of childbearing women is not so great as to constitute a contraindication for this form of therapy for condition in which it is seriously needed, but the potential danger is real enough to discourage irradiation when the indications for its use are not urgent or when the prospects of actual benefit are at all doubtful."

Case and Warthin have noticed changes in the liver indicating that general applications of radiotherapy, especially those over the abdomen, are apt to result in serious liver damage. It is entirely probable, they say, that postirradiation sickness is as much due to hepatic as to intestinal damage. They continue: "There is an additional general sensitiveness of some patients to radiation. Cachetic persons bear it badly, but in addition there are some persons who bear it badly although they have not developed cachexia."

Thus we see some of the dangers of the use of radiotherapy. While it may be claimed that these effects can be minimized by proper precautions, it must be apparent that we are dealing with agents whose powers for evil are not fully known and whose potential dangers are such that we must give them our earnest consideration before they are employed.

Another consideration of great importance is that radiotherapists are not always competent gynecologic diagnosticians. Even experts can make mistakes. Pregnancy may be mistaken for a fibroid tumor, extrauterine pregnancy for the same, while pus tubes, tumors, and

abscesses of the ovary, cancer of the sigmoid or rectum have all been mistaken and treated for fibroids. When an operation is done the results of mistakes are not so serious, for the true condition is discovered as soon as the abdomen is opened and before valuable time has been lost or any great harm been done. Accurate diagnosis is a prerequisite for the use of radiotherapy.

Some of the objections urged against surgery are the mortality of the operation, and the morbidity due to adhesions, hernias, and the dread of the operation. The mortality in the uncomplicated cases of fibroid tumors is practically nothing. During my work in Washington I can recall but two deaths following operations for these tumors. One was due to suppression of urine in a woman, one of whose kidneys had been destroyed by pressure of the large growth. The operation was very long and difficult. The other death was due to peritonitis caused by a pelvic infection present when we operated. It had been recognized and the operation was delayed several weeks. The patient was losing ground and the operation was done as a forlorn hope. I have not looked up the number of cases operated upon, but the mortality is a very small fraction of 1 per cent. Clark and Bock, in 267 cases of fibroid tumor operations, had a mortality of 0.7 per cent and in 169 uncomplicated cases the mortality was nil. They truly say that the mortality of operation in uncomplicated cases is rapidly approaching the vanishing point. Adhesions do result fairly frequently, but seldom do they rise to disagreeable symptoms. They can be largely avoided by careful suturing and gentle handling of the abdominal viscera during operation. Herniae should not occur. The dread of an operation certainly prevents a number of women from submitting to it even when it is urgently needed. This, in my opinion, causes more women to submit to radiotherapy than any one thing.

With this information at hand let us see which cases of fibroid tumors should be treated by surgery and which by radiotherapy. The cases to be treated surgically are large tumors, cases where a myomectomy can be done especially in pedunculated tumors, tumors in comparatively young women, submucous tumors, tumors complicated with other pelvic conditions requiring operation, rapidly growing tumors, tumors giving rise to pelvic pain, the presence of a marked secondary anemia not accounted for by uterine bleeding, and myomas in very nervous women. The cases in which radium or x-ray therapy is indicated are those women near the menopause who have profuse periods, where there are either no tumors present or where they are small, and where it has been demonstrated by curettage that carcinoma of the body of the uterus is not present. In borderline cases the presence of a serious lesion of the heart, lungs, kidneys, or a very pronounced anemia may decide in favor of radiotherapy. There are, however, very few cases of heart disease where operation is contraindicated.

The heart condition usually is markedly benefited by the removal of the tumor if it is of considerable size. Almost the same may be said regarding anemias. Most cases require operation and it is seldom that an anemia is so pronounced that an operation is contraindicated. I have operated upon patients with a hemoglobin of 20 per cent or less and have had no bad results. Indeed at times when a woman is flowing freely the operation, if an easy one, is the quickest and best way to stop the flow. I no longer regard a moderate anemia as a contraindication to operation.

In large tumors the menopause does not usually cause so great a shrinkage of the growth as to make us hope for amelioration of many of the symptoms produced by the tumor, such as a pressure on the ureters, bladder, intestines, blood vessels, the increased work thrown upon the heart, and the inconvenience caused by the size and weight of the tumor. The induction of the menopause may cause a certain amount of decrease in size of large growths, but it is too uncertain to be depended upon. In the early days of gynecology when operators were afraid to attempt the enucleation of these large tumors they practiced the removal of the ovaries, hoping thus to reduce their size. This practice has practically been abandoned. These large growths are prone to undergo degenerations, at times, malignant. They sometimes become infected and frequently cause omental and intestinal adhesions. Some think they cause degeneration of the heart muscle. Therefore, the removal of these tumors is strongly indicated.

In cases where a myomectomy can be done in comparatively young women it gives them a continuance of menstrual life and a hope of childbearing. Last year I delivered three young women upon whom I had done a myomectomy not in any case more than two years prior to the delivery. One was sterile and promptly conceived after the operation; one had a small fibroid in a previous pregnancy which grew to be a large one in a few years. The third woman had a myomectomy done during a pregnancy. She miscarried, but was delivered of a healthy child some eighteen months later. It seems to me that it is evident that in the young women a myomectomy or the removal of a portion of the whole of the body of the uterus is preferable to the sterilization of the woman with x-ray or radium. At times a portion of the uterine body, which allows menstruation, can be left. Even where the whole of the body of the uterus is removed the retention of the ovaries prevents the advent of the premature menopause with its disagreeable symptoms.

In submucous fibroid tumors it can be readily understood that the tumors are prone to undergo degeneration, infection, necrosis, etc. I have seen a few cases of complete inversion of the uterus caused by a submucous tumor. Radium or x-ray tends to increase the danger of

necrosis. The consensus of opinion of all writers is that these tumors should be removed

It is self-evident that, where one finds these tumors along with some other pelvic condition requiring a laparotomy, radiotherapy cannot be considered. It might be noted here that quite frequently in doing a laparotomy for a fibroid tumor, one discovers a tumor of the ovary or some other abnormality of the pelvic or abdominal viscera requiring operation. In a considerable number of cases where a hysterectomy has been done for a fibroid tumor, upon opening the cavity of the uterus one discovers a carcinoma of the body. In rapidly growing tumors the suggestion is sarcomatous degeneration, hemorrhage, necrosis, etc. Tumors which give rise to pelvic pain are often associated with chronic inflammation of the adnexa, or adhesions, and it is advisable to remove such growths. According to Clark and Keene a marked secondary anemia, not caused by hemorrhage, is suggestive of degeneration of the tumor and should be treated by removal of the growths.

In women nearing the menopause with small tumors where a curettage does not remedy the condition and does not reveal a carcinoma, radiotherapy is indicated. The strongest indication for its use is in those patients with adenomyoma of the rectovaginal septum where a removal of the growth is attended with great danger; the production of the menopause by x-ray, radium, or operation (the removal or destruction of the ovaries) will as a rule cause a cessation of the symptoms. I have had a case where x-ray applications gave apparently permanent relief. Where there are large tumors of the uterus or tumors of the ovaries accompanying the growth in the septum, operation is indicated.

My opinion then is, that in fibroid tumors of the uterus, surgery is the choice in the great majority of the cases. Where radiotherapy is used the diagnosis of the condition should be made by a competent gynecologist. Many women who have these tumors require no treatment. They should be under observation, however, and should there be evidence that the tumor is causing symptoms its removal is indicated. Since the mortality from operation is so small and the chances of trouble being caused by the tumors considerable, the tendency, on my part, is toward the enucleation of the tumor before it reaches a large size or causes pronounced symptoms.

In cancer of the uterus it is not so easy to differentiate between the cases in which one or the other method is indicated. The poor result of operation before the advent of the Wertheim method in cancer of the cervix and the high immediate mortality of this operation deterred many gynecologists from operating on these patients. The time consumed in doing the Wertheim operation is a factor in a trial of the substitution of x-ray or radium in their treatment. My experience in

operating on patients with cancer of the uterus has been such that in all cases hopeful of the complete removal of the growth I strongly believe that operation is the best treatment. My percentage of cures in cancer of the cervix operated upon is more than 40 per cent. In my private cases it is over 60 per cent. A number of the ward cases have been lost sight of and as these have been classed as failures it brings the percentage of cures much lower than it would otherwise be. The percentages of the cures by Wertheim, Peterson, Cobb, and Graves are respectively 42.5, 47.3, 57.1 and 34.1 per cent. The number of my Wertheim operations have not been large (not more than 25 to 30), but I have had no immediate mortality. I attribute this to my modification of it which lessens the loss of blood and shortens the time required to perform it. It seems to me that the present trend of opinion is toward operation in all hopeful cases. However, many believe that radium should supersede operation in cancer of the cervix.

A great many authors are apparently chary of expressing an opinion on the subject. A paper in *Radiology* by Burnam and Neill of the Kelly Clinic indicates that the views in this clinic have materially changed. In early cases of cancer of both cervix and body of the uterus they advise hysterectomy unless there is some strong contraindication. In borderline cases they advise hysterectomy when possible, the operation in both early and borderline cases to be followed by radiation in cancer of the body and preceded by it in cancer of the cervix. In inoperable cases they advise radium. They say that x-ray has no place in the treatment of these cases except as a palliative measure in the more advanced ones. Nearly all observers are agreed that hysterectomy is the treatment of cancer of the body of the uterus. So far as I can tell the present status of the question should be as follows: In all hopeful cases of cancer of the body of the uterus a complete hysterectomy should be done. The prognosis of these cases is very good, the majority of them being cured. In advanced cases there is no cure, and radiotherapy may be tried as a palliative procedure. In cancer of the cervix where the growth is apparently confined to the cervix the Wertheim operation should be done, followed by the application of radium. The very best statistics are those where this method is used. Scherer claimed that 48 out of 58 cases treated by this method were free from recurrences at the end of three years.

In borderline cases the same treatment is applicable unless the general condition of the patient is poor, when radium is preferable. In advanced cases of cancer of the cervix where the bladder is not involved radium is the treatment. One should not, however, delude his patient with a hope of cure; it fosters quackery on the part of the radiologist. Care should be taken in these cases that the application of radium is not excessive. I have seen several women whose suffering has been markedly exaggerated and whose lives were considerably

shortened apparently by the intensive use of radium. Marked bladder and rectal symptoms made them utterly miserable.

Some believe that in cancer of the cervix where an infection is present the application of radium unfavorably influences the infection and hastens the death of the woman by causing general infection or a cystitis and pyelitis.

In view of the fact that the application of radium or x-ray during pregnancy is apt to cause anomalies of development in the fetus, these agents should never be used in the treatment of uterine tumors in pregnant women.

In conclusion I would say that I think instead of advocating the abandonment of operative procedures in cases of cancer of the cervix our endeavor should be to so perfect the operative technic that more cases can be treated in this manner. It has not been shown that radium is superior to operation when the operator is skillful. Practically every case of cancer of the body of the uterus should be operated upon. In cases of fibroid tumors operation is the better treatment in nearly all cases where the services of a competent gynecologist can be secured.

1730 K Street.

A STUDY OF THE EFFECTS OF BLOOD TRANSFUSION IN OBSTETRIC AND GYNECOLOGIC CONDITIONS

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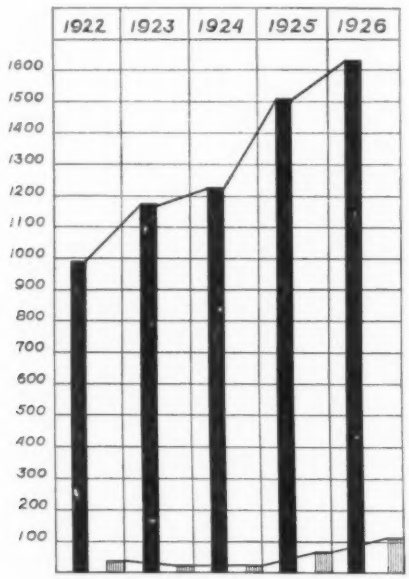
THE IMPORTANCE of direct blood transfusion as a therapeutic agent and as a life saving measure cannot be denied. It is therefore in an effort to arrive at a keener appreciation of some of its indications, limitations, and dangers, that this study was undertaken. For the result of our surgical audit must always be the basis for technical and therapeutic improvement as well as the indication for discard.

While it is not probable that this review will result in contributing anything new or startling to the subject of blood transfusion, we believe that we will be able to demonstrate that direct blood transfusion has a wider field of usefulness as a prophylactic and curative measure than any one remedy in our armamentarium; and that by its more general employment within the realm of obstetrics and gynecology, if the cases in which it is used are properly selected, the mortality and morbidity may be materially reduced.

This study is based upon 6539 admissions to the obstetric and gynecologic service of the Long Island College Hospital from January 1, 1922, to January 1, 1927, and necessarily includes patients suffering

from almost every type of acute or chronic lesion affecting the female generative system. Fig. 1 shows the gradual but constant increase in the number of admissions ranging from 922 in 1922 to 1632 in 1926, approximately an increase of about 125 patients a year. This increase in the census was only made possible, as there has been no addition to our bed capacity (the obstetric and gynecologic unit consists of 102 beds—public and private), by a greater turnover and a shortened convalescence. The factors which have contributed to this accomplishment are (1) *greater detail in the preopera-*



YEAR	CASES	TRANSFUSIONS
1922	992	33
1923	1176	20
1924	1229	20
1925	1510	67
1926	1632	116

Fig. 1.—Showing increase in census of Long Island College Hospital and incidence of transfusions.

tive care. Our staff has agreed in both clinic and private practice to use every available means for improving the general condition of the woman prior to having her admitted to the hospital for operation. This plan, of course, is limited to those applying for care who are suffering from subacute and chronic lesions such as the traumas of childbirth, pelvic inflammations, benign tumors, etc., in which time and local treatment have a beneficent effect. Such patients have their preoperative preparation done in the office and clinic and are

taught such rules of hygiene, rest and diet as will make them better operative risks. On the other hand, in those conditions in which the patient has sustained considerable blood loss, or is dehydrated, as may occur in ectopic pregnancy, placenta previa, ablatio, bleeding fibroids, etc., the individual resistance has been augmented by preoperative transfusion or transfusions and by intravenous infusions of glucose.

(2) At the time of operation every effort is made to minimize the amount of anesthetic used by preliminary "twilighting" with morphine and scopolamine, or if the amnesia and analgesia are sufficiently profound, the operation is done with local infiltration. Furthermore, an attempt is made to shorten the procedure as much as is consistent with good surgery, absolute hemostasis and the maintenance of perfect asepsis. All postoperative cases receive during the first twenty-four hours intravenous injections of glucose and from 1000 to 1500 c.c. of saline solution given subcutaneously.

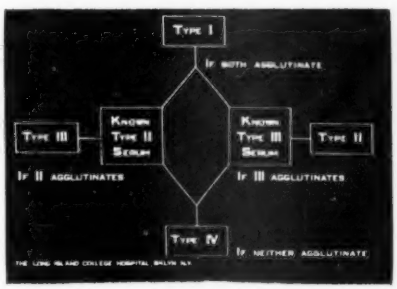


Fig. 2.—Diagram showing how matching may be simplified.

(3) By hastening the woman's recovery with fresh air, sunlight, and artificial light—we have found all of these agents raise the hemic content, increase the appetite and improve the woman's well-being. If, however, notwithstanding the employment of these agents the hemoglobin percentage remains below 50, the patient is transfused before leaving the hospital.

Methods of matching.—All of the transfusions given in this series have been grouped according to the Moss classification, and before any blood is given, it is rematched and cross matched directly with the cells and serum of the recipient. Each case has been grouped and matched by the interne and rechecked by the resident physician immediately before the transfusion was given. A schematic diagram (Fig. 2) shows how blood matching may be simplified.

Incidence.—By referring again to Fig. 1, it will be seen that the transfusions in 1922 show an incidence of one in every thirty patients; while in 1926 the relation is one transfusion in every fourteen admis-

sions. Prior to 1922 the citrated blood and the Lindermann multiple syringe methods of transfusion were used, but since that date each patient has received direct blood transfusion by the Unger or the Soresi technics. In all, 260 transfusions have been given, which would make an incidence of nearly 4 per cent, yet upon further analysis we find that these 260 transfusion operations were done on 164 different patients; the smallest number given to any patient was one, and the largest number of repeated transfusions was eleven.

Mortality.—Only one patient in this series died as the result of injecting blood. This woman should not have been transfused; her death was due to an error in election. She had a fibroid tumor of the uterus, which as a result of continued menorrhagia had produced a very severe secondary anemia complicated by a poorly compensated heart. She had the symptoms of decompensation a few days before receiving the transfusion—sufficient significance was not attached to this fact; and though but 200 c.c. were given, the load was too great for her myocardium—the right heart dilated and she died of a pulmonary edema.

Reaction.—There were no reactions to transfusion when the blood was given in cases of acute anemia, or where it was given as a prophylactic measure in the preoperative preparation of patients with a secondary anemia, or in the three cases of pernicious vomiting of pregnancy treated by blood transfusion. On the other hand, there were sixty cases or 23 per cent in which there were varying degrees of reaction. All of these patients were suffering from some form of infection, in none were the reactions particularly severe. Some few complained of chilliness, temperature rise and acceleration of pulse. These reactions cannot be attributed to any defect in matching, for all patients were grouped, matched, and finally cross matched directly with cells and serum of the recipient; and if on rematching, the serum of the recipient clumped any of the cells of the donor, the rule has been followed, "If in doubt, don't give it." Yet on repeated occasions we have found in septic cases, in some of the toxemias of pregnancy, and in the severe secondary anemias of splenic origin that the biochemistry changes from time to time and very speedily; for after grouping and matching, on the final cross match which is done immediately before the blood is given, it has been noted that the serum of the recipient will clump some of the cells of the donor. This was illustrated in one woman of Type 4, who was grouped and then directly matched with 12 donors and though they all belonged to Group 4, her serum clumped the cells of every one. In line with this observation we have the record of another interesting case: a woman suffering from a severe preeclamptic toxemia; this patient was of the same type as her husband and yet their bloods refused to match; later, however, after the baby had been delivered, her blood pressure

had been reduced and her condition improved, their bloods matched perfectly with no agglutination whatever.

These observations not only explain a large percentage of reactions met with in septic cases, but justify the dictum "if in doubt, don't give it"; and further demonstrate the value of the teaching that in all forms of sepsis and toxemia *the matching and rematching should be done immediately before the introduction of the blood*, as the biochemical changes in the serum and cell content are constantly changing with the variations in temperature, pulse and tissue oxidation.

Clinically we have found that when the transfusion was given during temperature remissions and when the injection was preceded by a hypodermic of morphine and scopolamine that the severity of the reaction was markedly reduced.

The size of the transfusions.—The average amount of blood injected has been 386 c.c.; the largest amount of blood introduced at one time was 800 c.c.; this was given to a case of acute anemia from hemorrhage due to placenta previa. Several small transfusions of 100 c.c. have been given and repeated in cases of infection with poor myocardia. On further analysis of these cases we find that 77 transfusions were done in the course of the treatment of infections during the acute stage. These include cases of postabortal fever, infected, incomplete abortions, puerperal metritis, parametritis, pelvic abscess, pyelitis, postoperative parotitis, thrombophlebitis and bacteremia. It is among these 77 that the large proportion of our reactions have occurred. Seventeen injections were given to make up for the blood loss in obstetric interpartum and postpartum bleeding; two were done to decrease the coagulation time in severe puberty hemorrhage resulting from endometrial hyperplasia, while the remaining 74 were employed for preoperative and postoperative conditions which required that the patient's resistance should be increased in order that she might be made a better surgical risk.

These conditions are illustrated in the menorrhagias and metrorrhagias of fibroid tumors which had produced a secondary anemia and that chain of tissue changes which occur as a result of a low hemic content, or in women recovering from long continued local infection with well defined foci ready for enucleation. These patients usually present a secondary anemia, low blood pressure, poor heart muscle and the general signs of asthenia. Transfusion in such cases changes the picture. Twenty-four of our cases fall in this class; there were four ectopic pregnancies in women entering the hospital with a hemoglobin percentage of less than 30 per cent who were transfused on the operating table. These women made good recoveries from their operation, yet in spite of feeding, sunshine and tonic treatment, they failed to regain their normal blood state. Postoperative transfusions in each of these patients resulted in a most happy change.

Hyperemesis of pregnancy.—The advantage of blood transfusion in the treatment of hyperemesis was an accidental observation with us some six years ago in dealing with a most intractable case of pernicious vomiting, who in spite of isolation, hypodermoclysis, corpora lutea injections and intravenous glucose infusions, continued to vomit and to go from bad to worse; her urinary output was diminished to 300 c.c. per day; her pulse rose to 140; the blood pressure fell to 80/50; there were acetone, diacetic acid and lucin bodies in the urine. On consultation it was decided that her only hope was evacuation, but her condition was so bad that we felt she would die on the table; so as a preoperative measure we transfused her with 400 c.c. of blood, and to my surprise within twenty-four hours the entire picture had changed. The pulse slowed, the pressure rose, her urinary output increased and the vomiting ceased.

In this series there were three women suffering from hyperemesis of pregnancy in whom intravenous glucose, subcutaneous infusion and proctolysis failed to raise blood pressure, to increase the urinary output and to check the vomiting. These women were transfused with 350 c.c. of blood without reaction and promptly ceased vomiting, increased their urinary output, raised their blood pressure and had their well being improved.

As a preoperative procedure.—Patients who have been ill a long time, with or without persistent blood loss, and show varying degrees of anemia, slow clotting time, and low blood pressure with high pulse rate, are improved by the introduction of fresh human blood. Illustrations of the type of patients in this series were women suffering from chronic pelvic inflammatory disease, submucous myomata, gall bladder disease with biliary obstruction, carcinoma of the ovary, carcinoma of the uterus, pyelonephritis, Banti's disease of the spleen, etc. All these women would certainly have died had the necessary surgery been undertaken without improving their surgical resistance by transfusion.

Observations on the effect of transfusion upon the blood picture.—When 300 c.c. of fresh whole blood has been introduced in nonseptic cases, the rise in the red cell count has averaged an increase of 481,266 cells. When, however, the woman was suffering from a blood stream infection, no rise was noted following the first transfusion; the pulse, however, has always been slowed and the blood pressure raised; unless the patient's condition has grown progressively worse, a second transfusion three or four days later has invariably raised the red cell count and the hemoglobin percentage. Rise in the latter has been greatest where the introduction of blood has been used to combat acute hemorrhage or as a prophylactic procedure in poor risk nonseptic cases. The average rise in all cases was 10 per cent. In some of

our septic groups the hemoglobin reading was even lower following transfusion than it was before the new blood was given. This has been accounted for in two ways: if the drop was slight (say, 1 or 2 points), it may be attributed to personal error in reading. On the other hand, when the drop has been considerable, it could usually be accounted for by the fact that in spite of the transfusion the patient's condition had become progressively worse. The effect on the white cells has been variable and has depended largely upon the clinical course of the disease or the condition for which the transfusion was given. The ratio between the leucocyte count and the polymorphonucluear percentage acted as a prognostic index in a number of cases. Where there was a continued rise in both white cells and "polys," the disease had usually become progressively worse. On the other hand, when the white cells showed a marked increase following the introduction of blood and the "polys" remained stationary or showed evidence of a drop, the clinical condition invariably showed coincident improvement; and when there was a drop in the white cell count to a leucopenia the prognosis was always bad.

We may draw therefore from this small series the following conclusions:

1. That the introduction of blood in properly selected cases is relatively safe, although it is not without danger.

2. That it raises the pressure, slows the pulse, increases the body functions, acts as a food, and stimulates the blood forming organs to the production of new blood cells.

3. That it has a wide field of usefulness in cases other than blood loss, particularly in increasing the resistance of the septic woman.

4. That as a preoperative agent it improves the resistance and makes the woman a better surgical risk.

5. That its effect on the blood picture except in acute hemorrhages is only that which results from improving body functions.

PYELITIS IN PREGNANCY

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ACUTE infections of the upper urinary tract have for many years been a serious complication of pregnancy. For want of a clear understanding regarding their etiology they were called "pyelitis of pregnancy."

Attention has been directed to the subject in the last two years by Hunner,[1] Kretschmer and Heaney,[2] J. O. Rush,[3] J. T. Williams,[4] and W. G. Sexton.[5]

During the past year and a half we have studied the cases of "pyelitis" during pregnancy which have occurred in the gynecologic-obstetric service at the Evanston Hospital. It was our custom to treat these cases by posture, free ingestion of water, and urinary antiseptics and, in case these methods failed to bring relief, by the ureteral catheter. In the great majority of cases these measures sufficed to bring temporary relief sufficiently to control the infection.

While nearly all the women were relieved sufficiently to make it possible for them to pass through pregnancy, it was frequently observed that patients who had a pyelitis during pregnancy were not entirely well after delivery.

Stimulated by the work of Hunner on stricture of the ureter as the predisposing cause of upper urinary tract infections, it occurred to us that the cause of the pyelitis occurring during pregnancy was already present before the pregnancy began, and that it was either a congenital or an acquired obstruction to the urinary outlet, instead of being due entirely to the pressure of the pregnant uterus, as is believed by some obstetricians and urologists at the present time.

With this idea in mind, we believe it is reasonable to assume that the obstructive pathology which existed during pregnancy, if due to obstruction rather than to the pregnant uterus, would be present after the uterus had been emptied.

It is not our purpose to take issue with anyone concerning the exciting organism or the route of infection, or whether the upper urinary tract during pregnancy undergoes a physiologic dilatation or not, but to report the pyelographic findings of cases of "pyelitis" occurring in pregnancy after the termination of the latter.

These investigations were carried out in the urologic department of the Evanston Hospital at periods varying from two weeks to four years after the termination of pregnancy, either by abortion or by delivery. They were made upon all women who had had "pyelitis" during pregnancy which was sufficiently severe to produce fever.

Pyeloureterograms were made in an effort to learn whether demonstrable changes in the urinary tract remained. The studies showed that in thirteen consecutive cases demonstrable distention of the kidney or ureter remained and obstruction of the ureter was present.

Therefore, the assertion which has so often been made, that the termination of pregnancy cures the pyelitis, is not wholly true. In these thirteen cases abnormal changes remained in the urinary tract after the termination of pregnancy and could be demonstrated as hydronephrosis, hydroureter, and ureteral stricture.

1. Mrs. S.—Six months after delivery.
   Right: Hydronephrosis, hydroureter, ureteral kink, ureterocele, stricture at ostium.
   Left: Double kidney, fused double ureter, hydronephrosis, ureteral kink, double hydroureter, stricture at ostium.
2. Mrs. M.—One year after delivery.
   Right: Beginning hydronephrosis, dilated ureter, ureteral kink, stricture of ureter.
3. Mrs. DeM.—Four months after labor.
   Right: Early hydronephrosis, ureteral kink, dilated ureter, ureteral stricture.
4. Mrs. Ha.—*A.* Four months after delivery.
   Hydronephrosis, dilated ureter, ureteral stricture at ostium.
   B. One year after delivery.
   Showing effect of treatment.
5. Mrs. A.—Four months after abortion.
   Left: Early hydronephrosis, ureteral kink, dilated ureter, stricture of ureter at ostium. Diagnosis by bulb.
6. Mrs. V.—Six months after delivery.
   Left: Hydronephrosis, ureteral kink, dilated ureter, ureteral stricture at ostium. Diagnosis by bulb.
7. Mrs. B.—Eight months after labor.
   Hydronephrosis, dilated ureter, ureteral stricture at ostium.
8. Mrs. I. S.—Three weeks after labor.
   Right: Hydronephrosis, ureteral kink, dilated ureter, ureteral stricture.
9. Mrs. Ho.—Two and a half years after delivery.
   Right: Beginning hydronephrosis, dilated ureter, ureteral stricture.
10. Mrs. S.—Two weeks after abortion.
    Right: Dilated ureter, ureteral stricture.
11. Mrs. K.—Four years after pregnancy.
    Right: Hydronephrosis, ureteral kink, dilated ureter, ureteral stricture.
12. Mrs. F.—Three months after labor.
    Right: Hydronephrosis, dilated ureter, stricture at ostium. Diagnosis by bulb.
13. Mrs. Sch.—Three months after labor.
    Right: Hydronephrosis, dilated ureter, ureteral stricture.

MANAGEMENT

Considering the frequent occurrence of pyelitis in pregnancy, one cannot but be impressed with the fact that the pressure of the pregnant uterus does play a rôle in urinary drainage during pregnancy. How great its influence is can only be determined by more careful and systematic observation of the woman in the early weeks.

We believe too little attention is given to the examination of the urine in pregnant women. Systematic catheterization with urine cultures should be done as a routine on all patients with a history of vesical irritability, either recent or remote, at least twice in the early months of pregnancy.

Pain referable to the urinary organs, a history of chronic appendicitis, either alone or associated with vesical irritability are indications for urologic study at this time and can be accomplished with little danger to the mother or fetus.

As pyelitis is secondary in a large group of cases to foci of infection in the teeth, tonsils, and sinuses, these regions must be cleared of infection as the first step. Then, if adequate drainage was provided for in the urinary tract, it would be interesting to see whether or not "pyelitis" occurred in the later months of pregnancy.

In all acute cases of urinary obstruction occurring early or late in pregnancy our plan is as follows: The patient is put to bed, a mild laxative given, and free ingestion of water encouraged, a glass every hour while the patient is awake. The patient is prepared for cystoscopy. A suppository of extract of opium and extract of belladonna, ¼ grain each, is given one hour before examination, together with ⅓ grain of pantopon intramuscularly one-half hour before. As both opium and pantopon lessen pain and relax unstriped muscle we have found this procedure sufficient to make a cystoscopy practically painless, at the same time lessening the spasm of the ureter that is always present after morphine is given.

Having passed, if possible, a No. 6 F. whistle-tip ureteral catheter to the renal pelvis on each side in order to ascertain from which kidney pus is coming, pelvic lavage is next performed on the affected side, using normal saline solution. This should be continued until the returned water is macroscopically clear. This procedure will insure better drainage while the indwelling catheter is in place.

While in place the catheter must be watched carefully lest it become plugged. It is a safe procedure to irrigate gently the renal pelvis morning and night with normal saline solution.

As the pelvic mucosa and ureter are already swollen and congested the application of antiseptics, we believe, only adds insult to the injury that already exists. Nature will take its course if free drainage is provided for.

As a rule there is marked improvement in symptoms in four or five days and the catheter can be removed. At this time it is safe to dilate the ureter gently by passing a No. 7 F. bougie. If there is a return of symptoms the catheter may be again passed and left in situ until the symptoms subside.

We believe forcible dilatation of the ureter to No. 9 F. or No. 10 F. at the first cystoscopy is a mistake. It is true that it gives ample drainage, the one thing desired in urinary infection, but at the same time it forcibly tears the mucosa and thus paves the way, by producing scar tissue, for additional obstruction in the future.

Gentleness is of the greatest importance in dilating ureteral obstructions. It is our custom always to lubricate the ureteral catheter or bougie with liquid alboline before any attempt is made to introduce it.

A ureter that is dilated to No. 9 F. or No. 10 F. will usually give sufficient drainage to permit pregnancy to continue to full term, but this is not true in all cases.

We do not believe a pyelographic study should be attempted as a routine until after the pregnancy has terminated. We wish however, to emphasize the fact that treatment of these cases is necessary after pregnancy ends.

Inasmuch as our cases have all shown definite changes in the urinary tract which could be demonstrated after pregnancy terminated we feel that it is fair to suppose that, in some cases at least, this pathology must have been present before the pregnancy began. The acute attack of urinary infection during the pregnancy, then, would be due to aggravation of the original lesion by the pregnancy, and possibly to some additional obstruction produced by the pregnant uterus.

We are greatly indebted to Drs. R. A. Scott, Charles E. Galloway, and E. C. McGill for permission to report their cases.

CONCLUSIONS

1. In thirteen consecutive cases upon which pyelographic studies were made after the termination of pregnancy pathology in the urinary tract was demonstrated.

2. The termination of pregnancy does not cure the urinary infection. These cases should be treated after the pregnancy ends in order to restore adequate urinary drainage, if possible.

3. As pathology of the urinary tract was shown to exist after termination of pregnancy in all cases studied, it seems reasonable to assume that obstructive pathology may have been present before the pregnancy began.

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THE STUDY OF STILLBIRTHS OCCURRING IN 4000 CONSECUTIVE DELIVERIES*

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UNLESS we are willing to review our records and study the results obtained, we are really not in a position to judge accurately, or to value rightly any line of treatment, or any form of surgical procedure. If we admit, as I am sure we all do, that growth in medicine is dependent upon the correction of mistakes as well as upon the adoption of new methods of treatment, then statistic studies are as valuable and as well worth while as research and experimental studies, and aid equally in the solution of many problems.

On an obstetric service there are three outstanding groups of statistics that register efficiency and good treatment in a most impartial and accurate way; namely, maternal mortality, infant mortality, and stillbirths. At the Woman's Hospital we have just completed a study of both the maternal mortality and the stillbirths, and we are still at work on that of infant mortality. These studies are all based on 4000 consecutive ward deliveries, covering seven and one-half years (June 26, 1919, to January 1, 1927).

I shall offer in this paper the results and figures obtained in the study of stillbirths, and I desire to consider especially the group of cases which might be termed remediable, those in which there was born a normal viable baby, and in which the fetal heart was heard during labor or at the onset of labor. It is the study of this group, I feel, that will give the most helpful information.

In these 4000 deliveries, there are classified as stillbirths all those cases in which the baby never breathed, even though the heart continued to beat after birth, and in which the delivery occurred between the twenty-eighth week of pregnancy and term. On this basis there are 105 stillborn babies, or 26 per 1000, but since one mother had twins, both born dead, there are only 104 patients to be considered.

*From the clinic of the Woman's Hospital.

In 55 of these patients no fetal heart was heard on admission to the hospital, in three there was doubt, while in 47 patients the heart was distinctly audible when the patient was admitted. Of these 47 there were 41 in which the heart was subsequently heard during labor, and 13 cases in which the heart continued to beat after the baby was delivered. Among the 105 stillborn babies 37 were premature and 68 at term; 44 were macerated and 61 were not.

In view of the amount of work that is being done to educate patients to the importance of prenatal care, it is very interesting to see that 85 of these mothers had been attending the prenatal clinic and only 19 were emergency cases and not admitted from the clinic, giving a ratio of stillbirths among clinic patients of 21 in 1000 deliveries. Further analysis shows that of the 58 cases in which a fetal heart was not heard or was doubtful on admission, 45, or 77.5 per cent, were from the prenatal clinic, while among the 47 in which a fetal heart was heard on admission there were 41, or 87 per cent, from the prenatal clinic. This series, though small, is further substantiation of the importance of antepartum care, showing a higher percentage of clinic patients among those who entered the hospital with living babies. At the Woman's Hospital, all patients are expected to report every two weeks to the clinic, and oftener, if necessary, thus placing a real responsibility upon the men in charge of this department, in recognizing and treating abnormal conditions. Clinic patients are admitted to the hospital ward whenever any condition arises needing hospital observation and care; and the records show that the most frequent cause for this is toxemia. The rule in the past has been to admit all women showing evidence of this condition, but recently we have made this rule more elastic and are now admitting arbitrarily every woman who has a prenatal systolic blood pressure of 140. This may seem rather extreme, but we hope our results will justify the extra hospital days required. In this group which we are studying there were 25 patients with evidences of toxemia and 79 without, but only 18 cases came from the prenatal clinic. Four of the 25 patients with toxemia had convulsions.

Negative Wassermann reactions were found in 70 mothers; nine were four plus. One patient had extensive condylomata lata, syphilitic in type, and the remaining 24 had no Wassermann tests made. These omissions, I may say, occurred early in the series. Thus there were ten cases of syphilis found among the 80 examined, or one in eight. To these ten luetic mothers two babies were born with lues, one having a marked pemphigus and one a four plus cord Wassermann; the remaining eight showed no signs of syphilis. All placentas are sent routinely to the laboratory for examination. In addition 23 babies had cord Wassermanns done. Of the 105 stillbirths 53 were

autopsied, and no other cases than those just mentioned gave any signs of syphilis.

A review of the autopsy findings on these 53 babies gives some interesting data, especially on intracranial injuries, of which there were 12. Three of these injuried babies were delivered by breech, five by breech following version, four by forceps, and one normally. In only one case was a fractured vertebra found; this occurred in conjunction with a torn tentorium and was not given as the cause of stillbirth. Table I is compiled from the reports of the pathologist.

TABLE I

Macerated Fetus		13
Cerebral Injury		12
Tear in Tentorium	7	
Tear in Falx Cerebi	1	
Tear in Falx Cerebi and Tentorium	1	
Cerebral Hemorrhage	3	
Intrauterine Asphyxia		7
Premature Fetus		4
Anencephalus		3
Hydrops Congenitalis		1
Fetus Papyraceus		1
Syphilis		1
Normal		11
		53

The cases of prematurity were not as frequent among these stillbirths as had been anticipated (Table II), there being only 37 babies born before term. These were very evenly distributed between the twenty-eighth and thirty-eighth weeks; 11 of them were in induced and 26 in spontaneous labors. In 14, toxemia was an associated condition; in 6, syphilis, and in 5, premature separation of the placenta.

TABLE II

28 to 38 weeks	6	34 to 36 weeks	7
30 to 32 weeks	7	36 to 38 weeks	10
32 to 34 weeks	7	38 to 40 weeks	68
			105

As one would expect, abnormal deliveries predominated, there being 61 (Table III), though it is surprising to find only 22 babies delivered with forceps. However, too much stress should not be laid on the method of delivery per se, when one considers that in more than half of the cases studied no fetal heart was heard on admission to the hospital.

TABLE III

Normal		44
Forceps		22
Low	5	
Medium	12	
High	5	
Breech		17
Breech after version		22
		105

Pelvic deformity was not an important factor in these cases, for ninety women had normal pelves (Table IV). Eleven were abnormal and three were not measured.

TABLE IV

Flat	6
Justo-minor flat	1
Masculine	4
Not measured	3
Normal	90
	104

Prolapsed cord occurred 11 times, in ten of which it was a contributing or actual cause of stillbirth.

Realizing that many figures confuse and are hard to assimilate, further study of these cases can be limited to the most important group, that in which a fetal heart was heard when the patient entered the hospital. If the baby was alive in 47 of these cases when the mother was admitted, and in 41 during part of her labor, why was there a stillbirth? This question is the one which should be carefully studied and all the important data tabulated in the hope that some definite and instructive conclusions may be reached. The six cases, in which a fetal heart was heard on admission, but not during labor (Table V), showed conditions which readily explain the stillbirths, and except for the two cases of prolapsed cord, could not have been altered. However, an earlier recognition in these two cases might possibly have helped in avoiding the stillbirths which resulted.

TABLE V

Eclampsia (emergency case, not from prenatal clinic. Labor induced).

Premature Labor (admitted with ruptured membranes at thirty-second week, but no pains. Autopsy: prematurity).

Abnormal Fetus (heart heard on admission, not afterwards. Autopsy: anencephalus).

Premature Separation of Placenta. (Labor induced. No fetal heart after insertion of bag. Hemorrhage 800 c.c.)

Prolapsed Cord—two cases (each admitted with history of ruptured membranes: in one, cord was prolapsed nine hours before onset of pains; in the other four and one-half hours. No pulsation in cords when found. One autopsy: normal baby).

The 41 cases in which a fetal heart was heard during labor show a variety of conditions, often several being associated in the same case. This has made it difficult to classify them concisely and at the same time to present tables that are accurate. However, there are certain outstanding points that can be briefly summarized, thereby enabling one to obtain quickly a general idea of this group of cases. Ten deliveries were spontaneous, 13 were with forceps, and 18 by breech, 14 of which followed version. This gives 75 per cent abnormal deliveries, with version and forceps about equal in frequency. Eight patients had a labor of over thirty-six hours and seven had ruptured membranes for more than twenty-four hours. There were ten persistent occipitoposterior positions, six being delivered by forceps after an instrumental rotation, and four by version and breech. Four presentations were breech, one was transverse, and one a face with chin posterior. Seven patients had deformed pelves, three being flat and four masculine in type. Three had a placenta previa, two a premature separation of the placenta, and five a toxemia. In seven babies the cord was around the neck, and in six the cord was prolapsed. Of the 18 babies autopsied, eight had a cerebral injury and ten were uninjured. It is interesting further to note that 31 of these stillbirths occurred in primiparae and ten in multiparae, or 75 per cent in patients having their first labor.

A more careful analysis of these 41 cases shows a group of 21 cases which may be called unavoidable. Either the complications were recognized early and all procedures that were possible instituted to meet them, or the stillbirths were beyond human control. Table VI shows these cases, listed according to the most important condition in each case.

TABLE VI

Prolapsed Cord	4
Prolapsed Arm and Cord	1
Placenta Previa	3
Persistent Occiput Posterior	3
(Medium forceps,—instrumental rotation.)	
Premature Separation of Placenta	2
Toxemia (induced labor)	2
Masculine Pelvis, medium forceps	1
Transverse Presentation	1
(With prolapsed arm.)	
Face Presentation, R.M.P.	1
Breech Presentation, dry labor	1
Cord around neck six times	1
Anencephalus	1
	21

There is a second group (Table VII) of eight cases in which the choice of a different type of delivery might have given the baby a

better chance. Seven were in cases with a persistent occipitoposterior position, three of which were delivered by high forceps and four by version and breech. One was an elective version with the head unengaged. These eight are borderline cases in which the choice of delivery was difficult, since the patients were often entitled to a test of labor (seven being primiparae). Though they form a very small percentage of the 4000 deliveries reviewed, they illustrate a most important point, if we are to make every effort to reduce stillbirths to a minimum. Looking back upon them with our present view of these problems, one must feel that the three high forceps deliveries should not have been done, and the choice should have been between cesarean section and version; also that the patient with two previous stillbirths (Case 4) should have had a cesarean section and not a version. On the other hand the patient with marked toxemia (Case 7) was probably best handled by a version, since, in our experience at least, toxic patients do not stand laparotomies well. In the remaining cases one may question whether the choice of a version was the best solution. However, as these were all primiparae much may be said in its favor. Prolonged labors are bound to occur, and if we decide upon a thorough test of labor, they are at times unavoidable. However, should we not seek in every way to curtail these? Personally, I feel that on a hospital service where a majority of the patients have in the prenatal clinic the advantage of frequent and careful observations, during the latter weeks of pregnancy, this group of borderline cases should be so well studied in the clinic, that after admission a decision can be made within a reasonable number of hours and without subjecting these patients and their babies to the risks of prolonged labors. These cases emphasize the importance of the most careful prenatal study in all cases where potential difficulties during labor or delivery exist.

TABLE VII

1. Primipara, at term, normal pelvis. Prolonged test of labor, lack of advance, persistent O.P. High forceps; prolapsed cord, caught by forceps blades. Baby 9 lb. 4 oz.
2. Primipara, at term, flat pelvis. Prolonged labor, bag for dilatation of cervix, persistent O.P. High forceps. Baby 7 lb. Autopsy: Cerebral hemorrhage. Subsequent delivery by cesarean section, normal baby.
3. Primipara, at term, flat pelvis. Prolonged dry labor, bag for dilatation of cervix, persistent O.P. High forceps. Baby 7 lb. 13 oz. Subsequent delivery by cesarean section, normal baby.
4. Multipara, at term, normal pelvis. History of two stillbirths at home, last one of which was 13 years previously. Mild toxemia, persistent O.P. Version and breech. Baby 7 lb. Autopsy: Bilateral tear in tentorium.
5. Primipara, at term, flat pelvis. Dry labor, persistent O.P. Version and breech. Baby 8 lb. 3 oz. Subsequent delivery by cesarean section, normal baby.

6. Primipara, at term, normal pelvis. Prolonged dry labor, bag for dilatation of cervix, persistent O.P. Version and breech, cord tight around neck twice. Baby 7 lb. 12 oz.
7. Primipara, at term, normal pelvis. Marked toxemia. Labor induced with bag. Persistent O.P., too high for forceps. Version and breech. Baby 7 lb. 9 oz. Autopsy: Torn tentorium.
8. Primipara, at term, normal pelvis. Prolonged test of labor, L.O.A.; too high for forceps. Version and breech. Baby 9 lb. 4 oz. Autopsy: Torn tentorium and fracture of 6th cervical vertebra.

The third group (Table VIII) contains 12 cases, being those in which a more careful watching might have resulted in the recognition of fetal embarrassment in time for a successful operative interference.

TABLE VIII

TYPE OF DELIVERY	NO. OF CASES
Spontaneous Delivery	
Uncomplicated (following normal labor of less than 13 hours. One autopsied: Asphyxia)	4
With cord around neck (following normal labor of less than 20 hours. Two autopsied: Asphyxia)	3
Forceps Delivery	
Low Forceps (following normal labor. Two autopsied: Asphyxia)	2
Medium Forceps (following prolonged labor. One autopsied: Cerebral hemorrhage)	2
(Following prolonged dry labor. Masculine pelvis)	1
	12

The importance of following the fetal heart rate carefully during labor is apparent, and the need of promptly recognizing fetal embarrassment is essential, if each baby is to be given every possible chance. At the Woman's Hospital we are constantly laying emphasis on this point. At the beginning of this series, in 1919, the interns and resident physician made the observations on the fetal heart. Soon, however, the nurses in the delivery room were taught to count this, and directed to report any changes noted. Then we required them to record their findings on the labor record hourly in the first stage and half-hourly in the second stage. At the present time, in addition to the observations made by the interns and the resident physician, the nurses count the fetal heart every half hour from the onset of labor, and when the membranes rupture, second stage contractions begin, or complications arise, they do this every fifteen minutes. Frequent observations are continued in the delivery room, on abnormal cases, and, where there is slow advance, on normal cases, for the unborn child is entitled to every protection until safely in the world. The practice of obstetrics requires most careful watching of both mother and baby throughout labor and delivery if the best results are to be obtained.

SUMMARY

This study of the stillbirths in 4000 consecutive ward deliveries occurring on the Obstetrical Division of the Woman's Hospital shows:

1. There were 104 patients who had 105 stillborn babies, or 26 stillbirths per 1000 deliveries.

2. There were 85 of these patients who had been attending the prenatal clinic, giving 21 stillbirths, among clinic patients, per 1000 deliveries.

3. There were 25 patients who showed evidences of toxemia, and four of these had convulsions.

4. Nine patients among the 79 who had Wassermanns were four plus. One showed clinical evidences of syphilis. To these luetic mothers two babies were born with syphilis. No other babies gave any signs of syphilis.

5. There were 61 abnormal deliveries, 22 being by forceps and 39 by breech.

6. There were 90 patients who had normal pelvic measurements.

7. There were 37 premature babies and 44 were macerated.

8. There occurred 11 cases of prolapsed cord in ten of which it was a contributory or actual cause of stillbirth.

9. There were 53 babies autopsied and 12 of these showed cerebral injury.

10. In 55 patients no fetal heart was heard on admission, and in three it was doubtful. The heart in 47 was heard on admission and in 41 of these subsequently during part or all of the labor.

11. An analysis of these 41 cases suggests that some of these stillbirths might have been avoided:

A. In eight cases if a different method of delivery had been chosen.

B. In 12 cases if an earlier operative interference had occurred.

121 EAST SIXTIETH STREET.

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